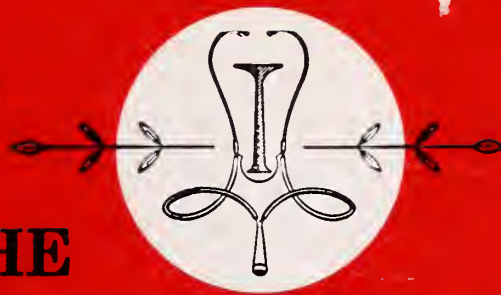


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The JOURNAL of the KANSAS MEDICAL SOCIETY

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Council Meetings

Report of Meeting Held September 18, 1983

The Council budget session convened September 18, 1983, in Topeka.

The Council adopted the following budgetary recommendations of the Executive Committee designed to significantly reduce expenses, increase revenues, and to balance the 1984 budget:

Salaries: No increases for 1984 to be considered at this time.

KaMPAC: 1984 grant frozen at \$4,000. KaMPAC Board encouraged to solicit funds for administrative expenses from clinics and professional associations.

Travel: Cut 20 per cent, saving approximately \$20,000. The following *out-of-state* travel will be allowed:

- AMA annual and mid-year House of Delegates meetings: AMA Delegates/Alternates and KMS President will receive coach airfare and \$150 per diem for each night out of state;
- AMA Leadership Conference: Coach airfare and expenses allowed;
- Other *out-of-state* travel will be by Executive Committee approval only.

In-state travel will be reimbursable to the President and staff only. All committee work by members will be performed on a voluntary basis.

Loans/Contributions: Approved the final contribution to Mediserve. No other loans or contributions to be allowed in 1984.

Auxiliary: Expenses limited to \$4,000, with recommendation that *Communique* be limited to two issues per year.

Journal: Additional \$5 per subscription (total \$15) allowed. Recommended continuation of the monthly publication; increase advertising rates; limit size — primarily in the scientific section — at the discretion of the Editorial Board to bring this budgetary item into alignment.

Dues: Increased \$20 in 1984 and \$20 in 1985. Additionally, KMS to look at outside sources of income.

In addition to the above actions related to budget matters, the Council also endorsed Future Innovations to sell AMA-GTE Computer Network service in Kansas.

Report of Meeting Held December 10, 1983

The Council convened December 10, 1983, at the Wichita Hilton Inn, for the purpose of reviewing interim committee reports. The following recommendations were approved.

Continuing Medical Education

1. To continue survey and accreditation of local organizations;
2. To continue publicizing educational programs to KMS membership; and
3. To budget appropriate expenses to continue recognition by ACCME.

KaMPAC

Educational plans for 1984 include presentations to component medical societies and hospital medical staffs. In addition, an AMPAC workshop is scheduled for March 1-2, 1984, in Topeka.

One of the major goals for 1984 is to secure more local input into the decisions on which local candidates to support.

Judicial Committee

KMS will pursue peer review of members and non-members for problems of ethics or medical competence.

Membership/Insurance Committee

1. Focus membership recruitment activities on students, residents, and non-member practicing physicians with the most potential.

2. Approved addition of partial disability benefits to disability income program sponsored by Washington National Insurance Company.

3. Study alternative to Blue Cross-Blue Shield for group health insurance. The following companies were contacted: Aetna, Hartford, SBL, Alliance, Bankers, Travelers. KMS members will be surveyed regarding their interest in an individual insurance plan while retaining Blue Cross-Blue Shield group coverage for those interested.

Committee on Health and Environment Liaison

1. Develop a joint statement with the Department of Health and Environment on long-term and aging care.
2. Develop a simple but precise survey about physician problems and concerns, and a prioritization for these concerns to be presented to the Department.
3. Make every effort to cooperate with the Department of Health and Environment in the revision of hospital rules and regulations.
4. Continue to serve as a liaison in making recommendations for H/E budget and departmental priorities.
5. Help develop methods of communication between the various segments of the Department and KMS members.
6. Develop a policy to aid H/E programs related to medical problems identified by the Department to act as a communications link, and assist when medical problems develop at the local level.

Committee for Impaired Physicians

1. The Kansas Medical Society must attempt to change the existing Kansas Reporting Law by providing an exemption for this Committee.
2. Until such time as the appropriate statutory changes are implemented, the function of this Committee will be limited to a policy-making, advisory body.
3. The Committee is asked to consider providing a reimbursement mechanism for time and mileage in cases referred through KMS for investigation and treatment.
4. Recommend that all referrals and specific arrangements for treatment should be made with individual physicians.

Allied Health Liaison Committee

1. Approve registration for: Occupational therapists, respiratory therapists, dieticians.
2. The committee recommends no change in the present status of physician's assistants.

Editorial Board

For the general report, see *The Journal of the Kansas Medical Society*, November 1983, page 545.

It is anticipated that these changes in association with judicious editing will bring and keep the *Journal* at the break-even point or better. However, the changes are not considered absolute and permanent. The Board will continue to function with due consideration for the economic necessities, but at the same

time seek to improve and expand the functions of the *Journal* as conditions permit.

SRS Advisory Committee

1. Move the entire Medicaid program to MediKan category, except for the justifiable cases of multiple medical conditions; Family planning to be provided in the physician's office; KMS to provide a committee to SRS to serve as an advisory panel to review medical necessity in cases on appeal. The committee will be appointed by KMS President and members will be reimbursed by SRS for transportation and per diem expenses; SRS to continue vigorous pursuit of fraud cases; If foregoing measures will produce greater savings than necessary to balance the budget, SRS will apply such overage to upgrade reimbursement rates on selected procedures which are now tied to the 50th percentile of 1975.

2. Physicians in Sedgwick, Saline, and Ottawa counties are advised that the role of KMS/SRS Advisory Committee in developing Primary Care Network program has been strictly in an advisory capacity. It is important for physicians to understand that participation in PCN is on a voluntary basis, and each contract must be reviewed and decided on an individual basis by the participating physician.

The choice of counties for the pilot project was made unilaterally by SRS. SRS is considering a contract with Bethel Clinic, Newton, for an HMO for pediatric services. Robert Harder, SRS Secretary, has stated that the Department will consider any offers for health care delivery in Kansas from any source.

Committee on Aging

1. KMS to provide physician coverage for the sessions of the Silver Haired Legislature;
2. Patient Information Brochures to be available to physicians and others at cost.

Kansas Medical Society Auxiliary

1. Continue work to increase membership;
2. Promote drug and alcohol abuse programs;
3. Promote forums on aging.

Kansas Hospital Association Liaison

1. Study standard guidelines for credentialing;
2. Continue liaison — confidentiality of records, medical staff section.

Legislative Committee

Awaiting final recommendations from KMS committees.

(Continued on page 24)



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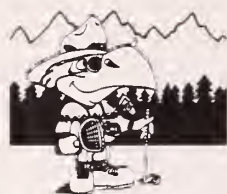


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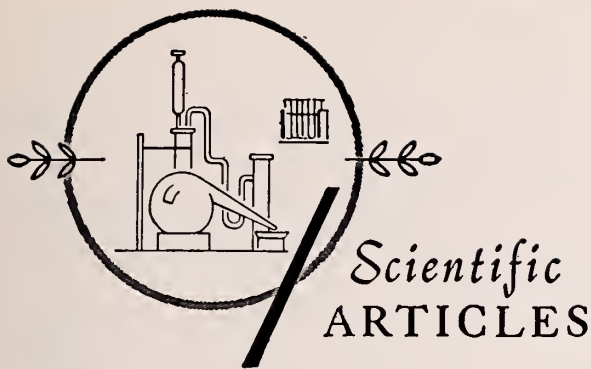
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Carcinomatous Meningitis

R. STEPHEN SMITH, M.D.*; JAY B. ZATZKIN, M.D.† and
HARRY E. HYNES, M.D., Ph.D.‡, *Wichita*

COLORECTAL carcinoma rarely metastasizes to the central nervous system. Metastatic involvement of the leptomeninges is an even more uncommon event. A review of the literature produced only four previously reported cases of carcinomatous meningitis secondary to colorectal carcinoma.¹⁻⁴ The plasma level of carcinoembryonic antigen (CEA) has been used widely as a tumor marker for colorectal and other malignancies.⁵ Experience with the significance of CEA levels in cerebrospinal fluid (CSF) is comparatively limited. Recent articles indicate that CEA levels in CSF may be used as an indicator of CNS involvement by malignancy.⁶ This may be particularly true for leptomeningeal metastasis.⁷

One case of widespread colorectal cancer with meningeal involvement as the only evident CNS disease in which both plasma and cerebrospinal fluid CEA levels were elevated has been reported.⁴ At our institution, a patient with carcinomatous meningitis presented one year after the initiation of therapy for adenocarcinoma of the sigmoid colon. Initial treatment had consisted of surgical resection followed by chemotherapy. At the time of cytologic diagnosis of meningeal disease, the cerebrospinal fluid CEA was found to be elevated while a simultaneous CEA level in the plasma was within normal limits. This appears

to be the first reported case of carcinomatous meningitis due to colon carcinoma with an elevated CSF CEA level and a simultaneous normal plasma CEA level.

Case Report

A 59-year-old Caucasian male presented with a three week history of insomnia and left lower extremity pain, and a three day history of diplopia, nausea, vomiting, headache, and hiccoughs. Twelve months prior to the onset of these symptoms the patient had undergone resection of an adenocarcinoma of the distal sigmoid colon with bladder invasion. Extensive nodal involvement was evident and could not be completely excised. No liver metastases were seen at the time of laparotomy. Surgical treatment was followed by monthly 5-FU infusions and an initial dose of mitomycin C with a favorable response. After eight months of chemotherapy, the 5-FU was withheld for two months due to persistent thrombocytopenia. During this period, retroperitoneal adenopathy began to reappear on computerized axial tomography of the abdomen, and plasma CEA levels rose. Following resumption of monthly 5-FU infusions, retroperitoneal adenopathy diminished, and plasma CEA levels returned to normal. It was at this time that the aforementioned neurological symptoms appeared.

Initial neurological examination revealed only disconjugate gaze. Subsequently, the patient rapidly developed deficits of the left III, VI, and VII cranial nerves, paralysis of both the left upper and lower extremities, and decreased sensorium. Computerized axial tomography of the brain was negative for mass lesions. Cytological examination of the CSF

From the Departments of Surgery and Medicine, The University of Kansas School of Medicine-Wichita; and St. Francis Regional Medical Center, Wichita.

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Address reprint requests to Dr. Smith, St. Francis Regional Medical Center, 929 No. St. Francis, Wichita KS 67214.

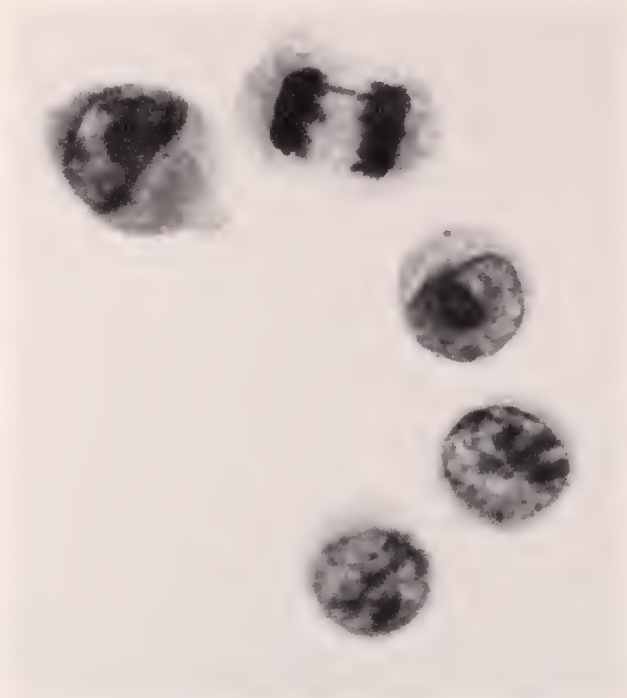


Figure 1. Cytologic examination of the cerebrospinal fluid revealed adenocarcinoma cells. Note the mitotic figure. (Papanicolaou $\times 400$)

showed adenocarcinoma cells consistent with gastrointestinal origin (Figure 1). The plasma CEA level was less than the upper limit of normal (1.8 mg/ml). Cerebrospinal fluid CEA levels obtained on two separate occasions were both elevated above the upper limits of normal for plasma CEA (6.4 mg/ml and 5.1 mg/ml). Lumbar puncture revealed an opening cerebrospinal fluid pressure of 155 mm H₂O. The CSF glucose level was 75 mg/dl while the protein level of the CSF was 140 mg/dl. The lumbar puncture was atraumatic with no red blood cells seen. The patient was immediately started on intravenous dexamethasone. Informed consent was obtained and the patient received 5-FUDR intrathecally via an Ommaya reservoir at a dosage of 2 mg/day for five consecutive days.⁸ Despite an initial improvement in results of laboratory tests with a decrease in protein and an increase in glucose in the CSF, the patient died one week following initiation of treatment, five weeks after the onset of symptoms.

Discussion

The incidence of carcinomatous meningitis secondary to colorectal carcinoma is quite low with only four cases previously reported in the literature.¹⁻⁴ Two recent large series of patients with carcinomatous meningitis have not included a colon primary.^{9, 10} These statistics are remarkable in light of the frequency of colorectal carcinoma in the general population.

The role of CSF CEA levels in the diagnosis of intradural metastatic disease is undefined. In 1975, Snitzer *et al.* first reported an increased level of CEA in the CSF of a patient with carcinomatous meningitis secondary to breast cancer.¹¹ Recent reports indicate that CSF CEA levels are independent of plasma CEA levels. Since evidence suggests that CEA does not cross the blood-brain barrier, the finding of an elevated level of CEA in the CSF should be a reliable indicator of intradural metastatic disease.^{6, 7, 12}

Conclusion

Carcinomatous meningitis secondary to colorectal cancer is rare but does occur. Of the four previously reported cases, the longest survival after onset of symptoms was four months. Present modes of treatment which include intrathecal chemotherapy and CNS irradiation have shown limited success in the therapy of this disease process. As longer survival of patients with colorectal carcinoma is achieved with the advent of more effective surgical and medical therapy, the incidence of carcinomatous meningitis could well increase. This trend has previously been observed in breast cancer, acute lymphocytic leukemia (ALL), and other malignancies.^{9, 12, 13}

In a patient with known colorectal carcinoma who develops neurological symptoms in the presence of normal computerized axial tomography of the brain, carcinomatous meningitis should be considered. In addition to evaluation of CSF cytology and chemistry, the CSF CEA may serve as an indicator of leptomeningeal involvement. The validity of CSF CEA levels as an indicator of leptomeningeal involvement is apparently unrelated to plasma CEA levels.

References are available from Dr. Smith, St. Francis Regional Medical Center, 929 No. St. Francis, Wichita KS 67214.

Chemonucleolysis

FORNEY W. FLEMING, M.D., *Wichita*

CHYMOPAPAIN, the major proteolytic component of *Carica papaya* latex, was first isolated and purified in 1941 by Jansen and Balls.¹ In 1956, Thomas reported the observation that rabbit's ears collapsed after intravenous chymopapain administration.² This report stimulated Smith to experiment with intradiscal injections in laboratory animals. He began clinical trials in humans in 1963 and coined the term "chemonucleolysis" to describe the procedure.³

Although early reports showed a favorable response in 70 to 80 per cent of cases,⁴⁻⁵ a double-blind study revealed no difference between intradiscal chymopapain injection and placebo injection.⁶ This report prompted the drug manufacturer (Travenol Laboratories, Inc.) to withdraw the distribution of chymopapain within the United States in 1975. However, the drug remained available in Canada and its widespread use continued there amid favorable reports of its effectiveness.⁷⁻⁸

In 1979 a new drug company (Smith Laboratories) was organized and began manufacturing chymopapain. A new double-blind study was undertaken, followed by an open-label study in the state of Illinois. The favorable results of these studies were evaluated by the FDA, and approval for intradiscal use of chymopapain in the United States was given in November, 1982.

Biochemical and Pharmacological Properties

In vitro studies indicated that chymopapain would cause hydrolysis of the nucleus of intervertebral discs with no apparent effects on the annulus fibrosis. Further investigation suggests that the primary action is to disrupt the water-binding properties of the mucopolysaccharide components of the nucleus, with no apparent effect upon collagen.⁹ This hydrolysis may account for the almost immediate relief of leg pain reported by some patients.

Toxicologic studies have shown a high margin of safety with the enzyme. High concentrations applied to dura, nerve tissue, ligaments, and bone do not appear to cause harmful effects. However, relatively small doses given interthecally can be toxic because of capillary rupture with resultant hemorrhage.¹⁰ Systemically, the enzyme has caused anaphylactic reaction in a small percentage of cases.

Address reprint requests to Dr. Fleming, 3243 E. Murdock, #200, Wichita KS 67208.

Indications

Chemonucleolysis is a surgical procedure which offers an alternative to discectomy. The patient's principal symptom must be radicular leg pain. The treatment is not indicated for localized back pain without radicular component. Physical findings should include positive straight leg raising with additional neurological abnormalities including weakness, parathesias, muscle atrophy, or diminished reflexes. Confirmation of a disc herniation is made by appropriate diagnostic studies which may include CAT scanning, myelography, and electromyography.

Before receiving chemonucleolysis, the patient should have an adequate trial of non-operative management. This would include at least two weeks of rest, physical therapy, and medication without improvement. An alternative approach would be three months of ambulatory care with restricted activity, rehabilitation exercises, medication, and supervised physical therapy.¹¹

Contraindications

The enzyme is currently approved for use only in the lumbar spine region. The major contraindications include:

- a known sensitivity to chymopapain or papaya
- progressive neurologic dysfunction
- spinal cord tumor
- cauda equina syndrome
- previous chymopapain injection
- spinal stenosis
- spinal deformity (*e.g.* spondylolisthesis)
- pregnancy

Additionally, poor results have been seen in the following cases:

- patients with previous surgery at the symptomatic level
- compensation cases

The reported risk of anaphylaxis is about 1 per cent. Any patient with a history of multiple allergies must be considered as an increased risk for an allergic reaction. This risk appears greatest in females with an elevated sedimentation rate.

The enzyme is not approved for use in children at this time.

(Continued on page 19)

Myoclonus Responsive to Vitamin B₁₂

WILLIAM J. NOWACK, M.D. and JENNIFER E. KENNEDY, M.D., Topeka

PARAMYOCLONUS MULTIPLEX — rapid, irregular muscular jerking of the arms and thighs — was so named by Friedreich in 1881¹ and attributed by him to increased excitability of the anterior horn cells.² As now termed, myoclonus is considered to arise any place in the nervous system and includes any muscular jerking, irregular or rhythmic. Although many etiologies are acknowledged, the case described here relates to metabolic disturbances not previously included.

Case Report

A 30-year-old obese, non-vegetarian white male was hospitalized with irregular jerking diagnosed as myoclonus. The onset occurred three years earlier, while the patient was in Navy service, as random twitching of the legs, left arm or face, and had been increasing. Cramping of the muscles and tingling of the areas were occasional accompaniments. Neurological examinations were repeatedly normal, and three-minute hyperventilation provocation produced no abnormality — specifically, no jerking.

Results of electroencephalography (during which some episodes of jerking occurred) and electromyography (with nerve conduction studies) were completely normal. Failure of quinine and aminophylline to alleviate the jerking led to a presumptive diagnosis of psychogenic jerking. The condition finally responded to diazepam.

The patient — now a civilian — was admitted for evaluation when the myoclonic jerking recurred after discontinuance of diazepam. Other than obesity (height, 168 cm; weight, 93.2 kg), results of physical and neurological examinations were again normal. There was no family history of neurologic disease, and no psychiatric disease was detected. Routine blood chemistries (including calcium determination), complete blood count, urinalysis, thyroid profile, erythrocyte sedimentation rate, antinuclear antibody, serum protein electrophoresis, serum immunoglobulins, and vitamin A (retinol) level were all within normal limits. Vitamin B₁₂ was 261 pg/

ml. Electroencephalogram showed a 10 Hz posterior predominant alpha rhythm and no significant abnormality. Schilling test showed normal absorption of vitamin B₁₂. Electromyogram of both lower extremities was normal. X-rays of the upper gastrointestinal tract, including both stomach and small intestine, showed no anatomical abnormality. Cervical spine and skull x-rays were normal; lumbosacral spine x-rays showed Grade II spondylolisthesis. The patient was begun on oral vitamin B₁₂ supplementation. Three months later, vitamin B₁₂ level was 410 pg/ml, and the myoclonic jerking was diminished markedly.

Discussion

The clinical description of the abnormal movements, with the normal electromyograms suggesting the absence of lower motor neuron or muscle disease, appears to satisfy the definition of myoclonus put forward by Marsden *et al.*³ The myoclonus appears to be related to the fluctuations in the vitamin B₁₂ level, although a placebo effect to explain the therapeutic efficacy of the vitamin therapy cannot be completely ruled out. The patient volunteered that while he was in the Navy, he was found to have a low level of vitamin B₁₂ and that his myoclonus had disappeared following supplementary vitamin treatment. Documentation of that assertion could not be found. The reason for the later presumption that the etiology was psychogenic was unclear. There was no evidence of an underlying psychiatric disturbance. This case emphasizes that a diagnosis of "psychogenic" should be made only upon finding of positive symptoms or signs suggesting psychological disease and not simply on the inability to find non-psychological pathology.

Some metabolic encephalopathies can lead to myoclonus.³ Encephalopathy is known to be one of the neurological complications of deficiency of vitamin B₁₂.⁴ However, deficiency of vitamin B₁₂ is not specified as one of the myoclonus-causing metabolic disturbances.^{3, 5} Patients with myoclonus secondary to metabolic disturbance described by Swanson *et al.* were more severely ill than the patient described above.⁵ Unlike those patients, our patient does satisfy the criteria for inclusion in the best prognostic
(Continued on page 19)

From the Colmery-O'Neil Veterans Administration Medical Center and Karl Menninger School of Psychiatry, Topeka.

Address reprint requests to Dr. Nowack, Neurology Service, Colmery-O'Neil Veterans Administration Medical Center, 2200 Gage Blvd., Topeka KS 66622.



Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Systemic Sclerosis

MARK E. PEARSON, M.D.* and DANIEL J. STECHSCHULTE, M.D.,†
Kansas City, Kansas

SYSTEMIC SCLEROSIS (scleroderma) is one of the three most common inflammatory rheumatologic diseases. Descriptions of the skin manifestations were first described in the 18th century but it was not until the mid 1900s that the generalized nature of the disease was widely appreciated. It is a systemic disease of unknown etiology involving internal organs as well as skin. Pathologically the disorder is characterized by diffuse inflammation, fibrosis, and vascular abnormalities.

Differential Diagnosis

Sclerodermatous syndromes include fasciitis with eosinophilia, localized (morphea) and linear forms of scleroderma which are not associated with systemic involvement. Fasciitis with eosinophilia is characterized by the rapid onset of swelling and pain, progressing to induration of the skin, subcutaneous tissue and fascia of the hands, forearms, feet and legs commonly associated with peripheral blood eosinophilia. Sclerodermatous syndromes with systemic involvement include systemic sclerosis and mixed connective tissue disease (MCTD). MCTD is an overlap syndrome of systemic sclerosis, systemic lupus erythematosus, and polymyositis. Differentiation of these two is important since some aspects of MCTD are responsive to steroids. Other rare forms of "pseudoscleroderma" which

have systemic involvement include vinyl chloride and silica exposure, acromegaly, primary amyloidosis, porphyria cutanea tarda, scleromyxedema, and the carcinoid syndrome.

Diagnostic criteria

Criteria for the diagnosis of systemic sclerosis were recently proposed by the American Rheumatism Association (*Table 1*). The major criterion (scleroderma proximal to the metacarpophalangeal or metatarsophalangeal joints) or two of the three minor criteria (sclerodactyly, digital pitting scars, bibasilar pulmonary fibrosis) are needed to confirm the diagnosis. These criteria have high sensitivity for the diagnosis and also are highly specific in separating systemic sclerosis from other rheumatologic diseases. Esophageal dysmotility is a common cause of symptoms in patients with systemic sclerosis but is not included among the diagnostic criteria as it lacks specificity.

TABLE 1
ACCURACY OF THE DIAGNOSTIC CRITERIA FOR
SYSTEMIC SCLEROSIS*

	<i>Sensitivity</i>	<i>Specificity</i>
Major Criterion		
—proximal scleroderma only	91%	99%
Major Criterion or 2 of 3		
Minor Criteria	97%	98%
—Sclerodactyly		
—Digital pitting scars		
—Bibasilar pulmonary fibrosis		

* Data from the Subcommittee for Scleroderma Criteria of the A.R.A. Diagnostic and Therapeutic Criteria Committee.

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Laboratory findings in systemic sclerosis include infrequent and minimal elevation of the erythrocyte sedimentation rate (ESR), mildly elevated IgG levels, low titer ($< 1:80$) rheumatoid factor (RF) in 30 per cent of patients, cryoglobulinemia in less than 5 per cent of patients, and a positive antinuclear antibody (ANA) by indirect immunofluorescence in 40-95 per cent of patients. Typically three ANA patterns are seen: speckled, which is the most common; nucleolar, which is the most specific for systemic sclerosis (seen in 20% of other rheumatologic diseases); and occasionally, a homogeneous pattern. Two other ANA patterns have recently been described using a human cell line as substrate. One type of autoantibody is associated with a discrete nuclear speckled pattern (anticentromere/kinetochore), and is selective for a subset of patients with calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia (CREST). Sera giving a diffusely grainy pattern of nucleoplasmic staining (anti-Scl-70) contain an autoantibody which may be specific for systemic sclerosis.

Clinical Features

Seventy per cent of patients with systemic sclerosis are female. The disease is most prevalent between the ages of 30 and 50 years and there is no racial preference. The usual complaints that bring the patient to a physician include Raynaud's phenomenon, swelling and puffiness of the hands, gradual thickening and tightening of the skin overlying the fingers, or diffuse polyarthralgia with joint stiffness.

Skin involvement is present in up to 95 per cent of patients. This occurs in three phases which provide some insight into the clinical stage of the disease. These include (1) the edematous phase characterized by painless pitting edema of the fingers and hands due to increased trapping of fluid. Occasionally the edema may be generalized. After several weeks to months this usually progresses to (2) the indurated phase caused by increased collagen accumulation resulting in thickened, firm, and shiny skin. Most patients remain in this phase, but a few may progress to (3) the atrophic phase characterized by a reduction of collagen content with thinning of the skin. Skin changes usually involve the fingers (sclerodactyly), hands, forearms, face, and trunk. In addition, capillary loop abnormalities in the nailfolds have been observed using low magnification microscopy. The severity of these changes has been shown to correlate with the degree of internal organ involvement.

Raynaud's phenomenon of the fingers, seen in approximately 90 per cent of patients with systemic sclerosis, is classically characterized by: pallor due to arterial constriction; cyanosis due to retardation of capillary blood flow; and rubor due to reactive hyperemia. Structural abnormalities of the vessel wall, present in patients with systemic sclerosis, predispose the digits to this low temperature and stress-induced phenomenon.

Gastrointestinal involvement, seen in 90 per cent of patients, usually consists of esophageal fibrosis and smooth muscle atrophy resulting in atonicity, dilatation, and loss of peristaltic activity. Many of these patients do not complain of dysphagia or reflux. Small bowel involvement is manifest by dilatation and stasis leading to bacterial overgrowth, deconjugation of bile salts, and a malabsorption-type syndrome. Rare features include pneumatosis intestinalis (air in the bowel wall) and large bowel wide mouth sacculations due to atrophy of the muscularis.

Lung involvement usually takes the form of interstitial fibrosis and has been found in most cases at autopsy. Pulmonary disease is second only to renal failure as a cause of death in patients with systemic sclerosis. Impaired diffusion capacity of the lung is an early measurable abnormality that precedes dyspnea. Patients may also have an increased incidence of alveolar cell carcinoma and adenocarcinoma. Pulmonary hypertension secondary to vasculitis is the most common cause of acute dyspnea.

Pericardial and myocardial heart disease can also be seen in patients with systemic sclerosis. Pericarditis is clinically present in 7 per cent of patients, but pericardial effusions have been detected in up to 41 per cent. Myocardial disease in the form of fibrosis and contraction band necrosis, may result in conduction disturbances. This is seen in 30-40 per cent of patients and is thought to be secondary to vasospasm of the coronary vessels.

Musculoskeletal disease is common and includes acute myositis or a chronic myopathy with atrophy. Resorption of the distal phalanges can be seen in the late stages of the disease due to an inadequate peripheral vascular supply. Symmetrical polyarthritides, seen in 10 per cent of patients, is less common than diffuse arthralgia with joint stiffness.

Kidney involvement is not as common (35%) as involvement of other organ systems, but oliguric renal failure and malignant hypertension are the leading causes of death in patients with systemic sclerosis. Patients with renal involvement may present with azotemia, proteinuria (usually greater than 500 mg/24 hr) and hypertension. Plasma renin levels are usually elevated before clinical evidence of renal

TABLE II
PATHOGENESIS OF SYSTEMIC SCLEROSIS

Possible defect in immunoregulation, resulting in autoantibody and lymphokine production
Vascular abnormalities
—Increased serum protease and esterase activity
—Circulating immune complexes
—Increased platelet activation and aggregation
Increased collagen accumulation

disease is present and may be a useful screening measure.

The CREST syndrome is a subset of systemic sclerosis with limited skin involvement, usually confined to the fingers and face. It is also thought to have a more benign course of internal organ involvement when compared to systemic sclerosis; in actuality, internal organ involvement does occur, but at a slower pace than typical systemic sclerosis, sometimes requiring decades to become evident.

Men with presentation of disease past the age of 45 years have more severe internal organ involvement than women. Early studies have shown an overall five year survival rate of 48 per cent and only 35 per cent survival after seven years. A recent study indicates a five year survival rate of 88 per cent in treated patients.

Pathogenesis

Evidence indicates that multiple defects are involved in the pathogenesis of this disease (*Table II*). Immunoregulatory dysfunction, such as a decrease in T suppressor cell activity or an increase in T helper cell activity, has been implicated. This may result in B cell production of autoantibodies and T cell production of lymphokines. These cell products may be responsible for the increase in collagen accumulation characteristic of systemic sclerosis. An increase in serum enzyme activity of proteases and esterases has been reported and has been implicated in endothelial cell cytotoxicity. Circulating immune complexes are present but their role in the disease process is not clear. Platelet activation and aggregation may also play a role and contribute to occlusion of vessels compromising blood supply. Ischemia and increased collagen accumulation in organ systems result in the clinical picture seen in systemic sclerosis. Although it is attractive to attribute these defects to a sequence of events initiated by an immunoregulatory dysfunction, additional information is required for complete understanding of the disease process.

TABLE III
DATA BASE FOR FOLLOWUP OF PATIENTS WITH SYSTEMIC SCLEROSIS

Physical Examination

- Upper to lower incisor measurement at maximal mouth opening
- Chest circumference at the nipples with maximal inspiration
- Degree of flexion contractures (mild, moderate, severe)
- Diagram showing the extent of skin involvement

Laboratory Tests

- ANA, ESR, CBC with differential, UA
- 24 hr urine for protein, creatinine clearance
- EKG, chest x-ray, pulmonary function tests (including diffusion capacity)
- Plasma renin level

Management

An initial thorough clinical evaluation including physical examination with objective measurements and laboratory data base are needed to manage and follow patients with systemic sclerosis (*Table III*). Office visits every three to six months permit serial objective measurements and repeating of laboratory tests as indicated by the patient's degree of organ involvement. The patient's subjective opinions are also crucial to assess success or failure of management. Frequent visits foster an appropriate patient-physician relationship, which is needed in this often discouraging disease.

Management of systemic sclerosis centers upon patient education and physical therapy (*Figure 1*). The patient should develop some insight into what is occurring pathologically in the skin and internal organs as well as the symptoms and findings associated with disease activity. Despite the frequent progression of the disease, some degree of optimism should be portrayed because therapeutic options are available, and thinning and softening of the skin can occur spontaneously. Physical therapy in the form of daily range of motion exercises, warm whirlpool, and massage is crucial during the active phase of the disease in order to preserve muscle strength and joint mobility as well as to decrease the degree of flexion contractures.

Symptoms and complications from disease activity are important management problems. Petrolatum or lanolin lubricant should be used to protect the skin, especially during exercise. Conservative management of Raynaud's phenomenon is often beneficial and includes avoidance of smoking and stress. Loose, but warm clothing and gloves should be worn if cold exposure is unavoidable. Aspirin in doses of

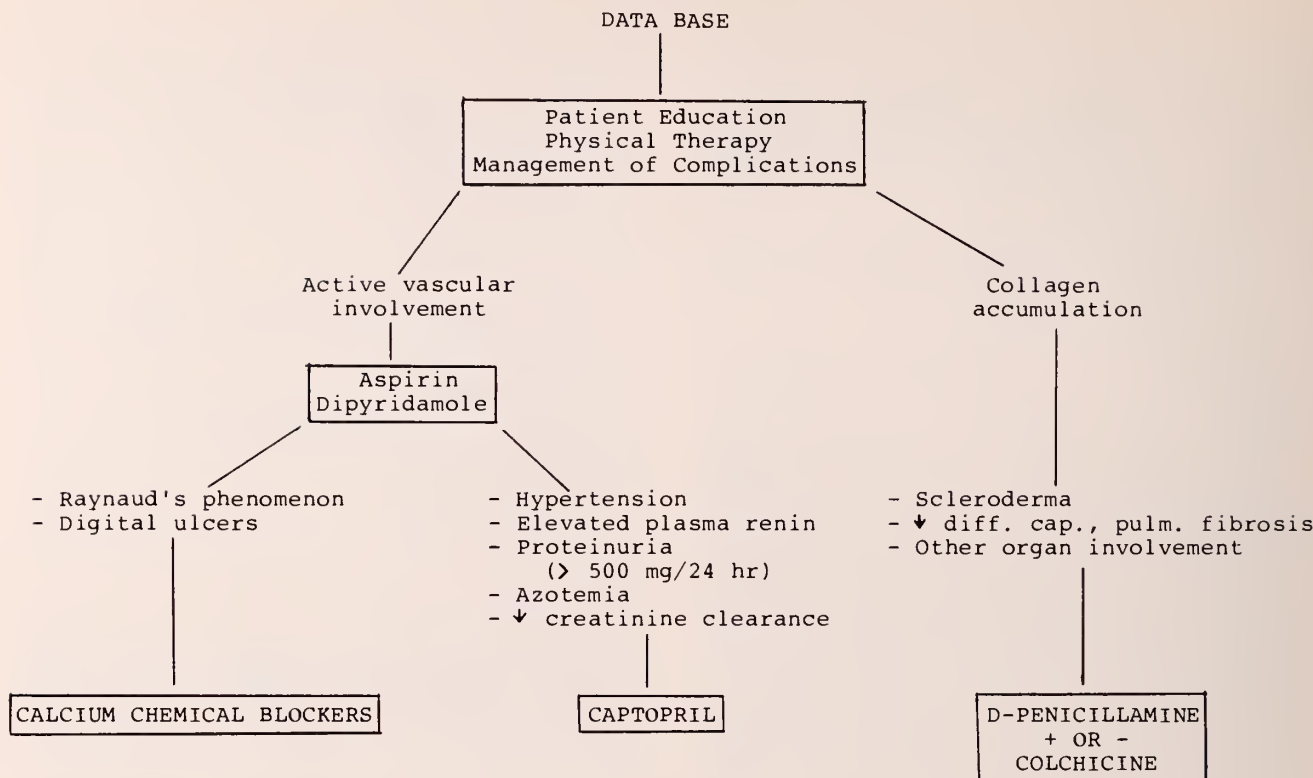


Figure 1. Flow Chart for the management of systemic sclerosis.

2.4-3.6 gm/day is helpful in obtaining symptomatic relief from arthralgias and joint stiffness, but renal function must be closely monitored. Esophageal reflux should be managed with antacids, elevation of the head of the bed, and the use of antagonists of histamine 2 receptors. Pulmonary infection or small bowel bacterial overgrowth warrant the use of broad spectrum antibiotics. Steroids, indicated for acute myositis with elevated muscle enzymes or isolated acute pulmonary hypertension secondary to vasculitis, appear to have little effect on the underlying disease process. Pericardial and myocardial involvement in patients with systemic sclerosis is managed routinely. Scleroderma renal crisis is an emergency situation; in the presence of severe hypertension, it requires the prompt and aggressive use of anti-hypertensive medications. Dialysis may be needed in instances of oliguric renal failure.

Pharmacologic Regimens

There are no documented controlled prospective studies that demonstrate efficacy of any pharmacologic regimen for the treatment of systemic sclerosis. Therefore, any currently recommended drug regimen must be considered experimental. However, there are selected case reports and retrospective

data which suggest that the disease process can be reversed and further progression prevented.

The rationale for the use of plasmapheresis and cytotoxic agents such as chlorambucil and cyclophosphamide is to correct the immunoregulatory defect. Cytotoxic agents have not been efficacious in the treatment of systemic sclerosis. Preliminary studies suggest that plasmapheresis, designed to remove the possible offending agent(s), has limited efficacy.

When there is evidence of impending or active collagen accumulation (*i.e.*, the edematous or indurated skin stages, decreased diffusion capacity, or other evidence of organ dysfunction), therapy with D-penicillamine is rational (*Table IV*). The objective and subjective response to this drug may be slow and subtle, mandating a three-month trial before increasing the dosage. The drug is discontinued with evidence of side effects.

A trial of colchicine may be warranted along with D-penicillamine if the disease is rapidly progressive or if an adequate response is not observed with 1000 mg or more of D-penicillamine. Colchicine at a dose of 0.6 mg 2 times/day orally has minimal side effects (possible gastrointestinal upset). Although studies so far have not shown the drug to be as effective as

TABLE IV
D-PENICILLAMINE THERAPY

Dosage
—begin at 250 mg p.o. daily
—↑ by 250 mg every 3 months until clinical response, toxicity, total daily dose of 1000-1500 mg
Side Effects
—Skin allergy (5%)
—Dyspepsia and taste disturbances (12%)
—Nephropathy (proteinuria and/or hematuria) (6%)
—↓ WBC, ↓ platelets (2-4%)
Monitoring
—UA and CBC with platelet count
—Initially, then every 2 weeks for first few months
—Then every 1-3 months thereafter

TABLE V
CAPTOPRIL THERAPY

Dosage
—25 to 75 mg p.o. t.i.d. (occasionally up to 600 mg/day)
Side Effects
—Proteinuria, mainly first 8 months (1-2%)
—Neutropenia/agranulocytosis first 3-12 weeks (< 1%)
—Hypotension
Monitoring
—Urine for protein initially, then monthly × 9 months, then periodically
—CBC with differential initially, then every 2 weeks × 3 months, then periodically

D-penicillamine, both medications do alter collagen metabolism *in vitro* and their synergistic effect is theoretically possible although unproven.

Raynaud's phenomenon is one manifestation of active vascular involvement in systemic sclerosis. The use of an alpha-adrenergic antagonist (tolazoline, phenoxybenzamine) or a calcium channel blocker (nifedipine 10-20 mg or diltiazem 30-60 mg 3 times/day orally) is effective in dilating cutaneous vessels of the extremities and has shown efficacy in the treatment of Raynaud's phenomenon with or without digital ulcerations. Although sympathectomy was once a common treatment modality, current indications are limited to intractable pain, severe ulcerations, or gangrene. The beneficial results of surgery are frequently transient.

Vascular abnormalities in systemic sclerosis may also involve the kidney. Preliminary studies using captopril (*Table V*) for the treatment of scleroderma renal crisis characterized by severe hypertension have shown drug efficacy. Since the drug acts by blocking the production of angiotensin II, the most potent vasoconstrictor known, a small oral dose (25 mg) may normalize blood pressure within one hour. Renal vascular involvement leads to an early elevation of plasma renin levels, usually before clinical evidence of renal disease (hypertension, decreased creatinine clearance, proteinuria, azotemia). An elevated plasma renin level may warrant usage of captopril in the hope of preventing further renal vascular

damage and development of clinical renal disease. If captopril and other antihypertensives fail to lower severe hypertension, bilateral nephrectomy with either transplantation or dialysis may be necessary. The drug also increases blood flow in the digital arteries with a decrease in symptoms due to Raynaud's phenomenon and healing of digital ulcers.

Anti-platelet therapy with dipyridamole 50 mg 3 times/day orally one hour before meals and aspirin 325 mg orally every other day has been used to counteract increased platelet activation and aggregation. The use of these medications with any evidence of active vascular involvement is suggested because of studies implicating platelets as a cause of vessel occlusion.

Current active research is directed toward understanding the pathogenesis of systemic sclerosis in order to find a management approach with greater efficacy and specificity. Controlled prospective studies are needed to further evaluate the pharmacologic regimens in use. The aggressive treatment of the complications of the disease, attention to patient education, and preservation of musculoskeletal function continue to be of paramount importance.

Acknowledgement

Max S. Allen, M.D., reviewed the manuscript.

A list of suggested readings is available from Dr. Stechschulte, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.



On Peering with Confidence

Since we are not involved in its production, we can express our unabashed admiration of the *Kansas Medical Society Newsletter*, in particular the 1983-7 issue which carried the report, "Confidentiality and Peer Review." The report relates, as readers will recall, to the action of the Kansas Supreme Court in disallowing the security of confidential communication to the proceedings of hospital peer review committees. In doing so, it declared that the administrative regulations relating to such activities did not have statutory authority to provide such protection. Moreover, the privilege of confidentiality rests not with the hospital or physicians but with the patient who thereby is the only one who can give consent to the disclosure.

In the process, the Court did strike a somewhat different note from some of the churlish comments other courts have addressed to the medical profession. It stated its confidence that the profession was a high-minded, selfless entity whose members would not be deterred in their actions or statements in peer review proceedings by the mere fact that the information could be made public. It complimented the profession on being "held in high esteem by the public and by the courts" which apparently should, by implication at least, endow such committees with adequate protection.

Grateful as we are for the Court's kind thoughts (and with, on the side, a thumbs-up signal to the Chief Justice whose lone dissenting opinion may take a little of the shine off the halo but comes much closer to the mark, we think), we have the disturbing feeling that we are working from different scenarios. We have gained the impression during the last few years that a considerable portion of the legal profession (and the public) takes a different view.

"Discovery" is what the lawyers call it, and it does need to be treated with respect since it works

both sides of the street. It means the other side gets to know everything you can't disguise and, even though equivalent opportunities are presumably available to you, it always seems to work out better for the other side. But it can hardly have escaped the justices' attention that peer review committees, working from their presently vulnerable state of legal authority, move into a much firmer risk of liability as soon as their comments and decisions are recorded. *Now* there is something to "discover" and legal ferrets will gladly dig it out and, if possible, disprove the Court's magnanimous opinion of the profession. And however virtuous the review effort or justified the action, there are few hospitals or physicians who do not have qualms about exposing themselves to such risks.

There is a certain irony about the Court's comments because that exalted state they assign to the profession is very close to the opinion it has of itself. Even the massive legal assaults of recent years have not changed this basic feeling, although they have produced much medical soul-searching, defensive maneuvering, and rearrangement of medical function. The problem relates, of course, to the fact that peer review committees are not protected by a specific statutory statement in Kansas — a situation in which the state finds itself reportedly in the company of only three others.

As the *Newsletter* indicates, the legislature will find the correction of this deficiency well up on its agenda at the behest of Kansas hospitals and of Kansas physicians who are now on record as being held in high esteem by no less an authority than the Supreme Court. We hope it will take precedence over the annual hassle with the Topeka City Commission over parking places or the perennial bid by the member from Bug Tussle to have the State Capitol moved there. — D.E.G.

Chemonucleolysis

(Continued from page 11)

Technique

The procedure should be performed in a hospital setting by physicians trained in the surgical management of disc disease, and who have had additional training in the use of chymopapain. In order to reduce the risks of an anaphylactic reaction the patient is begun on diphenhydramine (Benadryl) and cimetidine (Tagamet) 12-24 hours pre-operatively. The procedure may be performed under local or general anesthesia. After positioning the patient in the left lateral decubitus position, a routine back preparation is performed, followed by sterile draping. A C-arm fluoroscope is utilized for two-plane imaging, and a videodisc recorder is helpful to reduce total radiation exposure. Needle placement is performed via a lateral approach. Once needle placement in the center of the disc has been achieved, a discogram is performed to confirm proper positioning. Next, a test dose of 0.3 cc of chymopapain is administered. After waiting 15 minutes, the full dose of 1.5 cc (3,000 units) of enzyme is slowly injected. Multiple disc levels may be injected if indicated. The needles are carefully withdrawn and the patient is awakened. The patient is kept in the recovery room for two hours for observation since the risk of anaphylaxis is minimal after this time. Postoperative activities are determined by the patient's symptoms.

Summary

In properly selected patients, chemonucleolysis offers an alternative to surgical removal of a herniated lumbar disc. The success rate appears to be between 70 and 80 per cent, which compares favorably with discectomy.

Patients in whom chemonucleolysis has failed may still be satisfactorily treated with discectomy.

References are available from Dr. Fleming, 3243 E. Murdock, #200, Wichita KS 67208.

Myoclonus

(Continued from page 12)

group of patients with myoclonus — those with myoclonus but no seizures or other neurologic

deficit.² A syndrome including involuntary movements of the head, trunk, and limbs has been described in breast-fed Indian infants less than 1 year old who had markedly low vitamin B₁₂ levels (25-64 pg/ml) secondary to deficiency of vitamin B₁₂ in their mothers' milk. The disorder was responsive to small amounts of oral vitamin B₁₂. The infants had other problems, such as apathy, developmental regression and skin hyperpigmentation, which were not present in our case.^{4, 6, 7} Our case, which might be related to the syndrome described in the Indian infants, suggests that myoclonus can occur in the setting of a mild, subclinical vitamin deficiency in an otherwise well-appearing patient. This case suggests that the vitamin B₁₂ level should be determined in cases of myoclonus of unclear etiology.

Acknowledgement

George Monto, M.D., Ann Moore, and Lisa Aaron assisted in the evaluation of the patient and preparation of this material. Views are those of the authors and not of the Veterans Administration.

References are available from Dr. Nowack, Neurology Service, Colmery-O'Neil Veterans Administration Medical Center, 2200 Gage Blvd., Topeka KS 66622.

Vox Dux

Vox Dux Editor:

Concerning "The 1,000 Calorie Journal," I feel that the enormous amount of money wasted through KaMPAC would be better used to finance the JOURNAL. The numerous PAC groups are doing much harm to the political system which has worked so well to make this country great. Instead of a two-party system, there are now fighting splinter groups — none of which has much power, and none able to accomplish much except to make corrupt (in a way) politicians. I feel the political process would be much better served by *working in* and contributing to the political party of one's choice. Dedicated and knowledgeable elected officials confirm this. Too many splinter groups lead to anarchy.

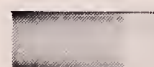
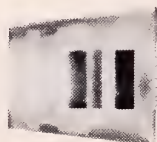
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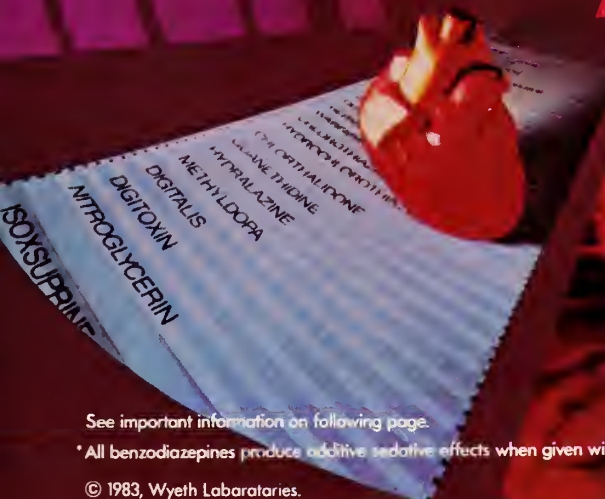
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Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid over-sedation. Terminate dosage gradually since abrupt withdrawal of any anti-anxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown, but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and micropthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chloridiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levaterenol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

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Jes Olesen, M.D., Hellerup, Denmark

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Council Meetings

(Continued from page 2)

Professional Liability Committee

1. Recommended developing a proposal to implement professional management and reporting of information concerning the HCSF, and appointment of a board of governors for review and approval of HCSF actions;

2. Recommended securing proposals from private companies to determine what types of coverage might be available and the costs involved if the HCSF were to be phased out;

3. Recommended studying the issue of tail coverage for resident physicians leaving the state and retiring physicians, perhaps establishing a minimum number of years of participation in the HCSF before the coverage would be purchased by the fund. Any time less than that would require individual purchase of the tail coverage by the resident or retiring physician;

4. Recommended studying the possibility of limiting coverage by the HCSF;

5. Recommended support of the following tort reform proposals:

- a limit on total awards
- a limit on pain and suffering
- abolishment of the collateral source rule
- limiting attorneys' fees by establishing a sliding scale mechanism
- reduction of the current judgment interest rate of 15 per cent to a more realistic level
- confidentiality of medical staff committee minutes
- requirement for itemization of a verdict
- establishment of a medical disciplinary account within the Board of Healing Arts
- allow periodic payments scheduled to expire on the death of the plaintiff

Opposition was recommended to establishment of prejudgment interest;

6. Recommended study of surcharges for various levels of claims against multiple claim providers;

7. Recommended that coverage provided by the Joint Underwriting Association be more realistically priced;

8. Approved a booklet informing physicians about malpractice prevention.

9. Proposed legislation may address the following points:

- Increasing the basic coverage limit from \$100/300,000 to \$200/600,000;

- Caps on liability at \$3 million with a \$6 million annual aggregate;
- Change the fund to an accrual accounting basis, amortizing the current debt over a five-year period, requiring a minimum surcharge of 25 per cent;
- Establishment of an advisory board to the Insurance Commissioner;
- Establishment of a mechanism to deal with multiple claim providers;
- Provision for confidentiality of peer review committees;
- Establishment of a disciplinary administrator within the Board of Healing Arts.

Other matters considered were as follows:

- *Blue Cross-Blue Shield Contract:* Recommend no action on this matter at this time.
- *Kansas Pharmacists Association:*

1. Disapproved Participation in a joint program to print state patient medication inserts. AMA PMIs are fulfilling current needs.

2. Disapproved investigating voluntary prior authorization agreements where physicians would be allowed to give standing orders, within agreed upon protocols, to pharmacists.

3. Approved formation of joint liaison committee to discuss prescription guidelines and other matters.

- *Advanced Registered Nurse Practitioners:* Referred to written comments of legal counsel. Authorized further court litigation if regulations are not substantially improved in the Legislature.
- *Prescription of amphetamines for weight control:* Reaffirmed the policy of opposition to any regulation or statute defining the use or misuse of a specific drug. Specific problems should be handled on an individual basis by the Board of Healing Arts.
- *Chiropractic:* The Board of Healing Arts ruled that chiropractors may use the term "chiropractic physician" and may perform school physical examinations. KMS petitioned the Board to reconsider its ruling. The Board agreed to table the decision until an Attorney General's opinion is rendered.
- *Medical Staff Section:* Recommended the establishment of a Medical Staff Section within the House of Delegates.

Complete minutes of both Council Meetings are available at the KMS office.

ONE

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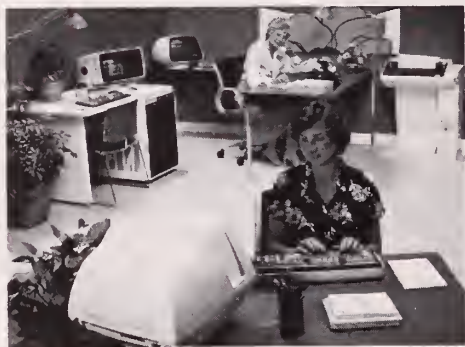
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- References:
1. Stone PH, Turri ZG, Muller JE. Efficacy of nifedipine therapy for refractory angina pectoris. *Am Heart J* 104 672-681, September 1982
2. Antman E, Muller J, Goldberg S, et al. Nifedipine therapy for coronary artery spasm. Experience in 127 patients. *N Engl J Med* 302 1269-1273, June 5, 1980

BRIEF SUMMARY PROCARDIA* (nifedipine) CAPSULES

For Oral Use

INDICATIONS AND USAGE 1. **Vasospastic Angina**: PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation; 2) angina or coronary artery spasm provoked by ergonovine; or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

2. **Chronic Stable Angina (Classical Effort-Associated Angina)**: PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS

Known hypersensitivity reaction to PROCARDIA
WARNINGS: Excessive Hypotension: Although in most patients the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug interactions: Beta-adrenergic blocking agents. (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates. PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy, Category C: Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients; transient hypotension in about 5%; palpitation in about 2%; and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72) and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59 to 77 F (15 to 25 C) in the manufacturer's original container.

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- 3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

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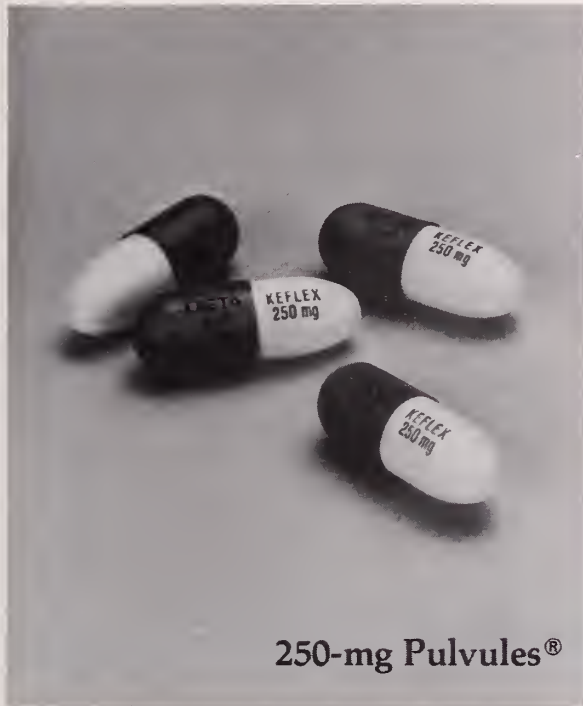
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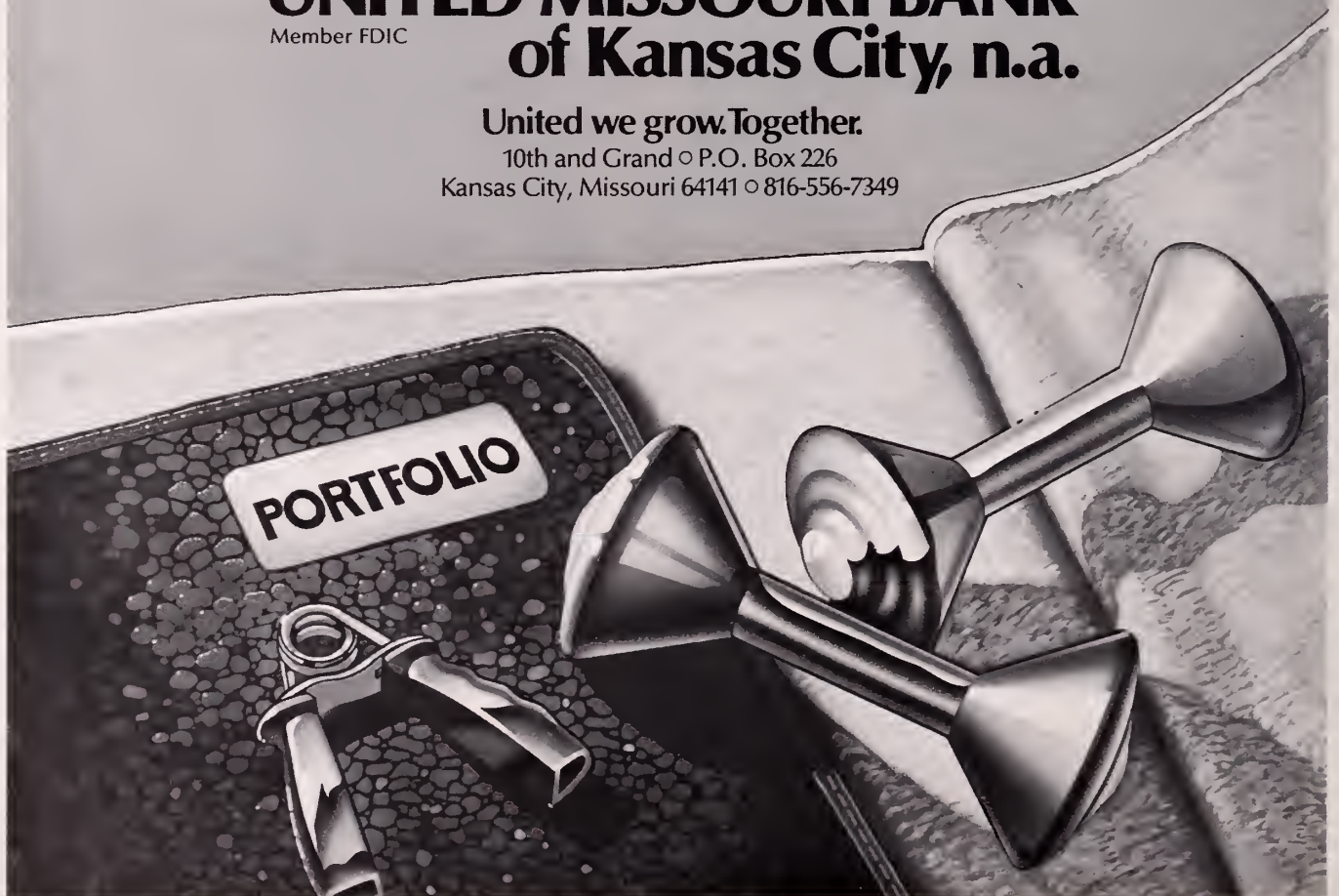
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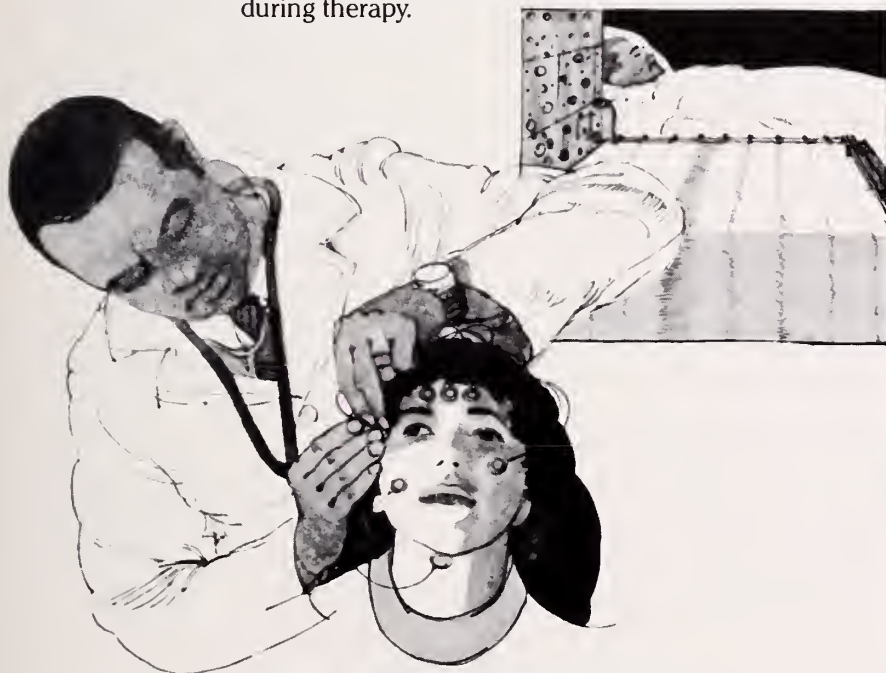
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References: 1. Kales A et al: *J Clin Pharmacol* 17:207-213, Apr 1977 and data on file, Hoffmann-La Roche Inc., Nutley, NJ. 2. Kales A: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. 3. Zimmerman AM: *Curr Ther Res* 13:18-22, Jan 1971. 4. Kales A et al: *JAMA* 241:1692-1695, Apr 20, 1979. 5. Kales A, Scharf MB, Kales JD: *Science* 201:1039-1041, Sep 15, 1978. 6. Kales A et al: *Clin Pharmacol Ther* 19:576-583, May 1976. 7. Kales A, Kales JD: *Pharmacol Physicians* 4:1-6, Sep 1970. 8. Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc* 27:541-546, Dec 1979. 9. Dement WC et al: *Behav Med* 5:25-31, Oct 1978. 10. Vogel GW: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. 11. Karacan I, Williams RL, Smith JR: The

sleep laboratory in the investigation of sleep and sleep disturbances. Scientific exhibit at the 124th annual meeting of the American Psychiatric Association, Washington, DC, May 3-7, 1971. 12. Pollak CP, McGregor PA, Weitzman ED: The effects of flurazepam on daytime sleep after acute sleep-wake cycle reversal. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, June 30-July 4, 1975. 13. Data on file, Hoffmann-La Roche Inc., Nutley, NJ.

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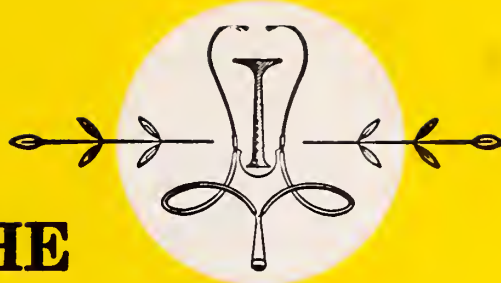
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The JOURNAL of the KANSAS MEDICAL SOCIETY

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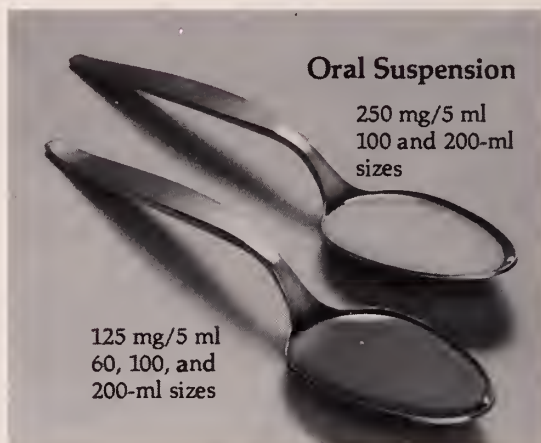
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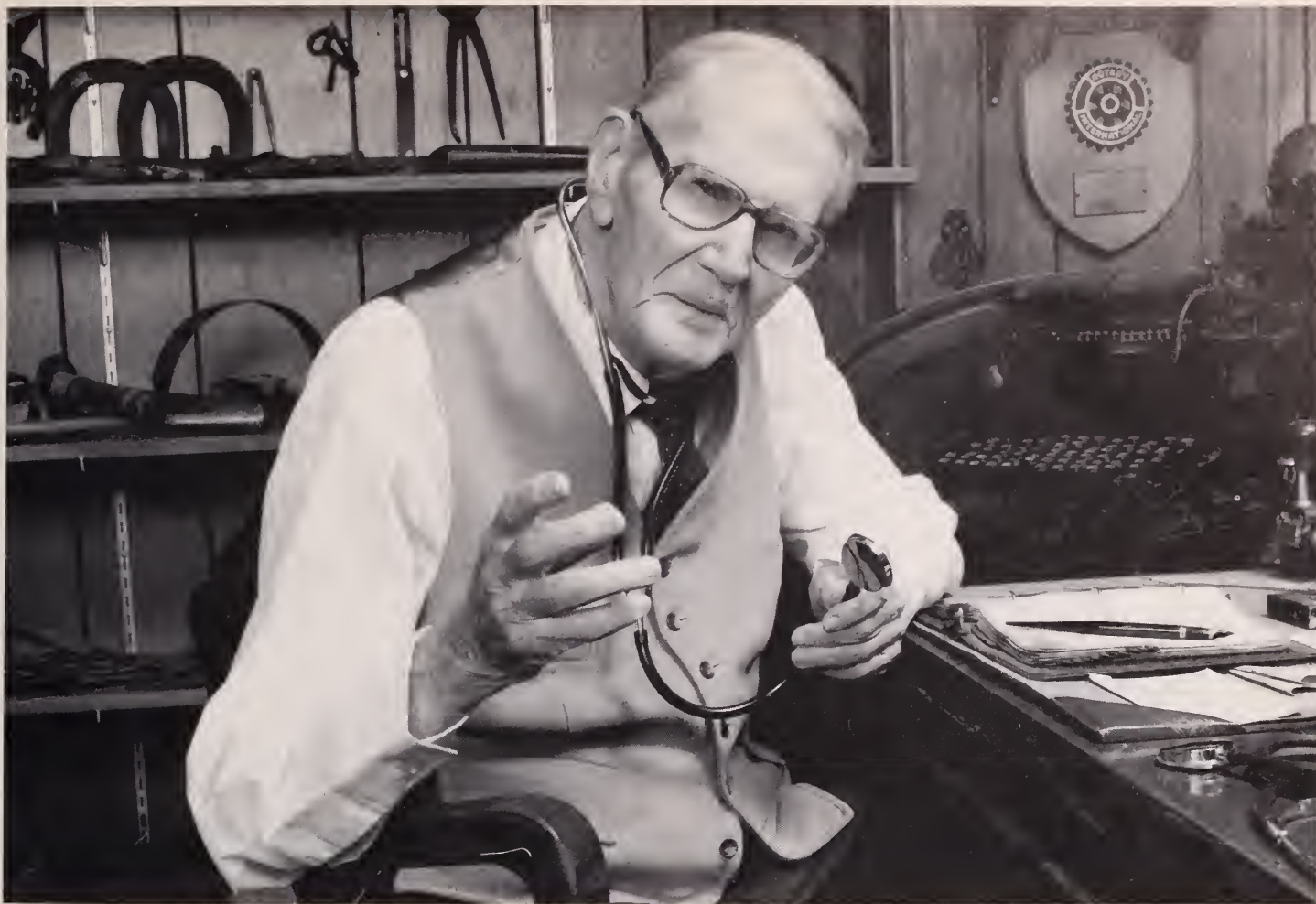


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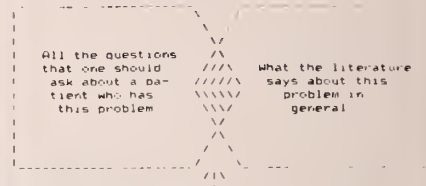
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Contraindications: Concomitant use with other potassium-sparing agents such as spironolactone or amiloride. Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids, and during concurrent use with amphotericin B or corticosteroids or corticotropin [ACTH]). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Cumulative effects of the drug may develop in patients with impaired renal function. Thiazides should be used with caution in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease. Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic and hemolytic anemia have been reported with thiazides. Thiazides may cause manifestation of latent diabetes mellitus. The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide; dosage adjustments may be necessary. Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported. Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Anti-hypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. Triamterene has been found in renal stones in association with the other usual calculus components. Therefore, 'Dyazide' should be used with caution in patients with histories of stone formation. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide'. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased dietary intake of potassium-rich foods. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function.

Thiazides may add to or potentiate the action of other antihypertensive drugs.

Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

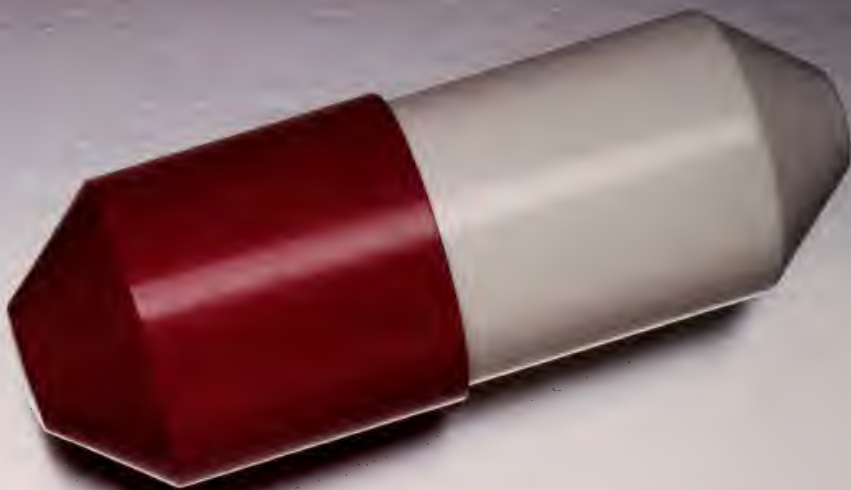
Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth, anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances; postural hypotension (may be aggravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and respiratory distress including pneumonitis and pulmonary edema, transient blurred vision, sialadenitis, and vertigo have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis have been reported. Impotence has been reported in a few patients on 'Dyazide', although a causal relationship has not been established.

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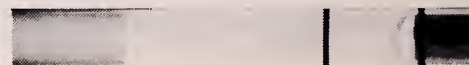
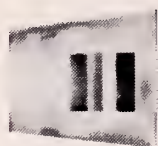
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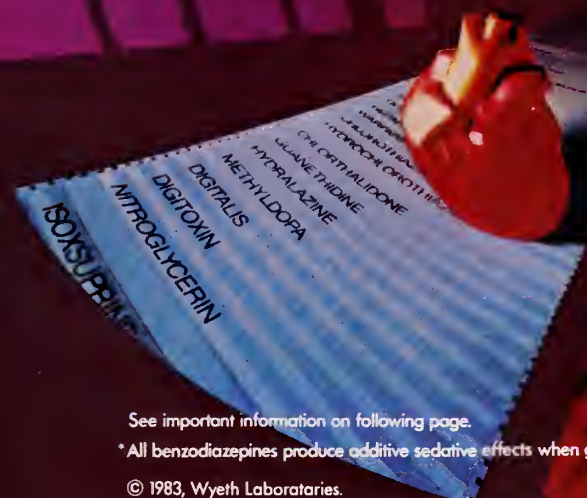
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Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic.

Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid overdosage. Terminate dosage gradually since abrupt withdrawal of any anxiolytic agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown, but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia, some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

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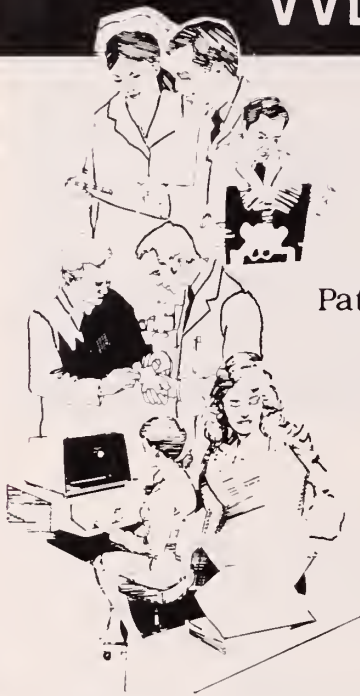
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Non-A, Non-B Hepatitis

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THREE TYPES of viral hepatitis are currently recognized: hepatitis A (infectious hepatitis); hepatitis B (serum hepatitis); and non-A, non-B hepatitis. While hepatitis A has been known in civilian (as epidemic jaundice) and military (as jaundice of campaigns) medical history for centuries, hepatitis B became prominent as the result of increased use of human blood and its products and frequency of various parenteral injections. Differentiation between these two types of hepatitis was made in 1926 by Flaum and his colleagues who recognized a short incubation jaundice (hepatitis A) and a long incubation jaundice (hepatitis B) among patients treated at a diabetic clinic in Stockholm, Sweden.¹ Subsequently the etiological agents of both hepatitis A and hepatitis B were characterized. The agent of hepatitis A is a picornavirus (a small RNA virus) and is now classified as Enterovirus type 72; the agent of hepatitis B is the first member of a new family of DNA viruses and is now classified as Hepadnavirus type 1. Moreover, laboratory procedures for the specific diagnosis of these two diseases and for the detection of the causative agent of hepatitis B in donated bloods have been developed and are now in general use throughout the world. However, in mid-1970s, when serologic screening tests ostensibly prevented the use of hepatitis-transmitting bloods, many cases of post-transfusion hepatitis still occurred which could not be linked to either hepatitis A or hepatitis B agents. Moreover, laboratory tests for the involvement of Epstein-Barr virus and cytomegalovirus were also negative in these cases. Thus a new type (or types) of hepatitis was recog-

nized; it was initially referred to as hepatitis C but now is designated as non-A, non-B (NANB) hepatitis.² Presented here is an update of information on this third type of hepatitis.

Epidemiology

Multiple episodes of viral hepatitis in drug addicts, suggesting the involvement of more than two etiological agents, have been observed since mid-1950s. Furthermore, reports published since 1975 have indicated that the majority (currently about 90%) of transfusion-associated hepatitis cases are of the NANB type. A study of sporadic cases of acute viral hepatitis in Baltimore, Maryland during February 1979-August 1980, indicated that 42 per cent were NANB hepatitis. Various risk factors were associated with 78.1 per cent of these cases; these were parenteral drug use (41.7%), ingestion of raw shellfish (12.5%), blood transfusion (11.5%), direct patient care or hospital laboratory work (6.3%), and intimate contact with others who had had hepatitis within the previous two to six months (6.1%). The remainder of cases (21.9%) had no identifiable risk factor.³ The 42 per cent frequency of NANB hepatitis among sporadic cases, studied in the above-mentioned geographic area, is higher than those previously reported for hepatitis cases in other areas of the United States which averaged about 25 per cent. Elsewhere, the frequency of NANB hepatitis in sporadic cases of viral hepatitis, has been generally lower than 20 per cent; *e.g.*, 10 per cent in Costa Rica, 13 per cent in Britain, and 14 per cent in Denmark. In Japan, however, about 50 per cent of clinical cases of hepatitis are classified as NANB type. Moreover, the frequency of this type of hepatitis is higher in hospitalized patients as compared to

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cases detected in a community survey which includes both hospitalized and non-hospitalized patients. The relative importance of various risk factors in the transmission of NANB hepatitis also varies with different study groups and different geographic locations. In contrast to the above-mentioned Baltimore study, a West London community survey indicated that drug addiction and administration of blood and its products did not constitute important factors in the transmission of this disease. Skin puncture (vaccination, inoculation, and dental treatment) and close contact were the important factors.⁴ As NANB hepatitis is now considered to be worldwide in distribution, additional epidemiological features are emerging. Many cases have been detected among hemophiliacs, hemodialysis patients, renal-transplant recipients, burn patients, donors in a plasmapheresis center (for production of anti-D plasma), and institutionalized mentally retarded persons. Moreover, in one epidemic of NANB hepatitis, the transplacental transmission of the agent was documented.^{2, 5, 6}

Etiology

The etiological agent (or agents) of NANB hepatitis has not yet been characterized. The first electron micrograph of candidate NANB hepatitis virus particles was published in June 1978. The aggregated particles, measuring 27 nm in diameter, were observed by immune electron microscopy in an homogenate of a liver biopsy obtained from a chimpanzee inoculated with a commercially produced NANB hepatitis-causing antihemophilic factor VIII. The inoculated chimpanzee developed enzymatic and histopathologic changes consistent with a diagnosis of viral hepatitis.⁷ However, differences in clinical and epidemiological features of NANB hepatitis as well as the occurrence of multiple attacks of this type of hepatitis indicated the possibility that more than one etiological virus may be involved. In 1979, chimpanzees inoculated with plasma derived from two different cases of NANB hepatitis developed two different ultrastructural alterations in their liver cells indicating the involvement of two viruses. In one group of animals, peculiar tubular structures, composed of two unit membranes, with electron-opaque material in between, were observed in the cytoplasm. In the other, only nuclear changes usually associated with aggregates of 20-27 nm particles — resembling viruses but not uniform in structure — were seen.⁸ However, subsequent investigations indicated that the nuclear changes were nonspecific and most likely due to the cytoplasmic changes. Moreover, four chimpanzees which had

been inoculated with plasma, serum, and a concentrate of factor VIII (all three implicated in human cases of NANB hepatitis), and had subsequently recovered from NANB hepatitis episodes, were cross-challenged with different inocula. In three animals, a second episode of NANB hepatitis developed which indicated the presence of two NANB hepatitis viruses (either two serotypes or two distinct agents).⁹ However, in a similar study in which serum lots, obtained from three chronically infected humans (residing in three different geographic areas; *i.e.*, Georgia, District of Columbia, and Connecticut), were used for primary and cross challenge infections of three chimpanzees, no recognizable NANB hepatitis developed after the challenge.¹⁰ This suggested that one agent or a group of antigenically related agents is involved in the etiology of this type of hepatitis. As to the morphology of the causative viruses, electron microscopic studies performed in the United States, Britain, France, Federal Republic of Germany, Belgium, and Japan have shown particles of different structures (*i.e.* with or without envelopes), locations (intranuclear, intracytoplasmic, or in the plasma), and sizes. These particles were 27 nm (in liver homogenate), 27 nm (intranuclear), 25-30 nm (intracytoplasmic), 32 nm (enveloped with an inner core of 22 nm in the serum), 35-40 nm Dane particle-like (in the serum), and 60 nm (enveloped with an inner core of 40 nm in the serum). The finding of Dane particle-like virus in certain NANB hepatitis sera has led Prince and his colleagues in the United States and Trepo and his colleagues in France to speculate that one common type of NANB hepatitis may be caused by a member of the hepatitis B virus group, which lacks hepatitis B surface antigen but shares hepatitis B e antigen and hepatitis core antigen.^{11, 12} A recent study showed that the NANB hepatitis virus transmitted by blood transfusion is not excluded from the cryoprecipitate prepared from plasma of symptomless chronic carriers and may also be present when such an infectious cryoprecipitate is processed further into antihemophilic factor VIII concentrate.¹³ Another study showed that the documented infectivity of a human serum was inactivated by treatment with formalin in a concentration of 1:1,000 at 37 C for 96 hours.¹⁴

Pathogenesis and Pathology

The pathogenesis and pathology of NANB hepatitis have features of both hepatitis A and hepatitis B. Although the route of infection is predominantly parenteral, infections acquired through ingestion and close contact are not uncommon in the developed countries. The incubation period ranges

from 35 to 70 days but may be as short as 14 days or as long as 120 days. Viremia develops prior to clinical illness and lasts for months to years after recovery. Infected human plasmas from chronic cases generally contain 10^2 to 10^3 infectious doses per ml for chimpanzees; however, in one acute case, the titer was at least 10^6 . Elevation of alanine aminotransferase (ALT, formerly SGOT) is observed for one to six months or longer. The so-called epidemic NANB hepatitis which occurs in parts of Asia and the Middle East and also among Europeans returning from these areas, is an acute self-limiting disease which afflicts predominantly young adults. However, it is more severe in pregnant women especially during the last trimester. The epidemic disease is acquired by ingestion of contaminated substances, especially water, and is spread through a fecal-oral route. As in hepatitis A, most individuals in the endemic areas are infected with the epidemic NANB hepatitis agent early in life and a significant percentage of cases are asymptomatic. Attempts to transmit this agent to chimpanzees have been largely unsuccessful. A recent study of this disease in the eastern USSR suggested that the causative agent may be a distinct but previously unrecognized serotype of hepatitis A virus.²

NANB hepatitis agents have been successfully transmitted to chimpanzees. Moreover, there is evidence that indicates that NANB hepatitis interferes with hepatitis A and B virus infections in chimpanzees. In these animals, the agents produce an acute hepatitis which may progress to a chronic disease. Histopathologically, certain characteristic changes occur in the liver cells of patients and experimentally infected animals. Light microscopy reveals cytolytic lesions with minimal inflammatory cell infiltration. Generally there is focal degeneration and necrosis of hepatocytes with cytoplasmic acidophilic bodies and accumulation of predominantly mononuclear cells. Electron microscopy shows disruption of hepatic endoplasmic reticulum into tubular structures in the cytoplasm of affected cells. Moreover, nuclear degeneration (which may or may not be disease-specific) generally associated with viral particles, ranging in size from 27 to 60 nm, has also been observed. These histopathological changes are often temporally associated with the acute disease and elevated ALT.¹⁵

The acute post-transfusion NANB hepatitis commonly (in about 40-60% of cases) progresses to chronic liver disease characterized by fluctuating ALT level and recurrent hepatitis. In chronic active hepatitis, a significant periportal reaction, with accumulation of mononuclear cells leading to fibro-

sis with tendency to septal proliferation and erosion of the limiting plates, and loss of individual cells in the peripheral parenchyma (piecemeal necrosis) are observed. Bridging necrosis, the landmark of hepatitis B, is absent in these cases; only about 10 per cent of these cases progress to cirrhosis.¹⁶ As biochemical evidence of hepatitis tends to diminish after one or more years, the long-term prognosis in these patients is unknown. This type of chronic liver disease is not common in epidemic (waterborne) NANB hepatitis.¹⁷

Clinical Features

These are generally similar to those of hepatitis B; however, NANB hepatitis is a milder disease, and only about 20 per cent of patients become jaundiced. Moreover, a significant percentage of epidemic NANB hepatitis cases are asymptomatic. The onset is usually insidious, and the usual symptoms of nausea, vomiting, anorexia, malaise, and fatigue are observed. ALT level is significantly higher than the normal level (*i.e.*, 40 IU/L) at onset and a range of 400 to 2000 units is detected during the course of the disease. One case of cryptogenic NANB hepatitis complicated by a hypocomplementemic serum sickness-like syndrome (rash and arthralgia) associated with circulating IgM immune complexes and developing to fatal aplastic anemia was recently reported. This syndrome occurs in as many as 30 per cent of hepatitis B patients but infrequently in hepatitis A cases.¹⁸

Laboratory Diagnosis

No unequivocally reproducible and universally acceptable laboratory procedure for the diagnosis of NANB hepatitis has yet been developed. As the etiological agent (or agents) of this type of hepatitis has not been yet isolated and characterized, the diagnosis is made by excluding hepatitis A and hepatitis B as well as infection with Epstein-Barr virus or cytomegalovirus. In 1978 a positive double immunodiffusion test between acute-phase sera (containing antigen) and convalescent-phase sera (containing antibody) from post-transfusion NANB hepatitis patients was reported.¹⁹ Subsequently, a similar test utilizing counterelectrophoresis procedure with acute and convalescent sera from experimentally infected chimpanzees and naturally infected humans was reported. Moreover both indirect and direct immunofluorescent tests with convalescent sera were reported²⁰ which apparently detected specific antigens in the hepatocyte nuclei of infected chimpanzees and humans. However, a more recent investigation of the antigen involved in the immuno-

diffusion tests revealed that it is not specific for NANB hepatitis.²¹ As immune complexes are detected in the sera of NANB hepatitis patients, they may mask viral antigens and thus prevent the conventional immunological reactions. However, another recent report described the specificity of an immunodiffusion test, applied to the sera of 26 NANB hepatitis patients (19 acute and 7 chronic persistent), which showed a single antigen-antibody system. The markers were not detected in sera from patients with immune complex or other liver diseases, or in healthy blood donors without a history of hepatitis.²² The above findings must be confirmed and extended by other investigators before a standard specific diagnostic test can be universally accepted.

An enzyme-linked immunosorbent assay (ELISA) using an antigen designated DS-Ag (derived from hemophilia A patients repeatedly treated with commercial blood products) and sera from convalescing post-transfusion NANB hepatitis patients was recently reported. The test was positive in 65 per cent of these patients, and reacted with neither hepatitis A or B virus antigens nor cytomegalovirus or Epstein-Barr virus. Moreover, a DS-Ag containing serum caused a typical NANB hepatitis in a chimpanzee.²³

Prevention

As there is still no reliable serological test for NANB hepatitis, screening of blood donors for the detection of the agents of this disease is currently unfeasible. The relationship between elevated ALT levels (see above) in donor bloods and the incidence of NANB hepatitis in the recipients of such bloods was recently established. Three different studies of the efficacy and economic considerations of such a screening, based on elevated ALT (>45 IU) in all blood donors, have been conducted. Members of the Transfusion-Transmitted Viruses Study (TTVS) of the National Heart, Lung and Blood Institute, concluded that 40 per cent of NANB post-transfusion hepatitis could have been prevented by discarding blood units with an ALT level of >45 IU. However, about 60 per cent of patients receiving bloods with ALT levels above 45 IU, did not develop hepatitis while 5 per cent of patients who received bloods with a normal ALT level developed the disease. Thus an elevated ALT is obviously not always a specific marker for the carrier state. Moreover, the so-called

"transaminitis" patients could not be permanently rejected as blood donors since the ALT test would eliminate 10 to 20 times more donors as compared to hepatitis B surface antigen test. As regards the potential economic benefits of excluding all bloods with elevated ALT, no definitive policy decision can be made at this time because only broad benefit estimates are now available and there are major uncertainties about the medical consequences of this type of hepatitis.²⁴ However, a recent communication from the Missouri-Illinois Regional Red Cross advocated the immediate initiation of ALT testing.²⁵

Addendum in Proof

Evidence for a virus in the fecal-oral route transmission of NANB hepatitis (see text) in a human volunteer has now been obtained.²⁶ NANB hepatitis has also been transmitted experimentally to Tamarins.²⁷ Preexisting, unresolved or current and acute NANB hepatitis has been shown to interfere effectively with the replication of hepatitis B virus in chimpanzees and thus the appearance of the latter disease is markedly delayed and moderated.²⁸ Evidence of NANB hepatitis obtained by an immunofluorescent test (using an antigen-antibody system linked to this disease) in six of 11 children with acute leukemia and chronic liver disease, has been reported.²⁹ The DS-antigen (see text) was shown to be a complex substance related to NANB hepatitis with no resemblance to particles connected with types A and B hepatitis or other known pathogens.³⁰ The specificity of a new immunoprecipitin test for NANB hepatitis in Costa Rican patients has been described.³¹ All infectivity of a plasma containing at least 10^4 chimpanzee infective doses of NANB hepatitis agent was destroyed by treatment with chloroform.³² Treatment with pepsin, pasteurization (60C for 10 hrs) in sorbitol-glycin solution and cold sterilization by beta-propiolactone combined with ultraviolet irradiation have been proposed to prevent transmission of NANB hepatitis by intravenous immunoglobulin.³³

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References are available from Dr. Behbehani, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.

Restrictive Cardiomyopathy in Scleroderma

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PROGRESSIVE SYSTEMIC sclerosis is a connective tissue disease which may affect the heart by conduction disturbances,¹ myocarditis,² focal myocardial lesions,³ abnormalities of the pericardium,⁴ pulmonary hypertension with associated dysfunction of the right ventricle, angina, and myocardial infarction with normal coronary arteries,⁵ and congestive and restrictive cardiomyopathies.⁶

A patient with previously undiagnosed scleroderma presented with pericardial effusion and restrictive cardiomyopathy.

Case Report

A 66-year-old white male was hospitalized for evaluation of easy fatigability and a 17-pound weight loss during the preceding six months. Diagnosis of diabetes mellitus had been made six months earlier, and the patient had been treated with acetohexamide with good control of blood sugar. Physical examination disclosed no abnormalities except mild underweight and a grade I/VI systolic murmur. Laboratory studies revealed: hemoglobin, 13.4 g/dl; hematocrit, 40.5%; MCV, 83; WBC, 7550 with segs 77%, lymphs 20%, eos 3%; sedimentation rate, 29 mm/hr; BUN, 14 mg/dl; creatinine, 0.8 mg/dl; albumin, 3.6 g/dl; total protein, 6.1 g/dl; and additional normal findings. Also normal were upper gastrointestinal tract, barium enema, intravenous pyelogram, and chest x-rays, sigmoidoscopy, and bone scan.

On readmission six weeks later, the patient had increasing fatigue, dyspnea, orthopnea, and pitting edema of the lower extremities. System review disclosed a history of tightness of the skin of his hands for about six months. No complaints of chest pain, Raynaud's phenomenon, or gastro-intestinal symptoms were elicited.

On examination, blood pressure was 168/90; pulse, 100; respiration, 20; with no pulsus paradoxicus. There was tightness of the skin of the hands and fingers. There was jugular venous distension, but no Kussmaul's sign. Chest examination revealed bibasilar rales and dullness to percussion. Heart auscultation disclosed a grade I/IV systolic murmur and an S₃. There was 2+ pitting edema of both lower extremities. No other abnormalities were noted.

Laboratory findings included: hemoglobin, 9.9 gm/dl; hematocrit, 30.5%; MCV, 82; WBC, 9100 with segs 76%, lymphs 11%, monos 6%, eos 6%, bands 1%; platelets 234,000, reticulocytes 4.2%; and Westergren sedimentation rate, 44 mm/h. Urinalysis was normal except for a trace of protein. The serum chemistry was normal except for: albumin, 3.4 g/dl; BUN, 38 mg/dl; creatinine, 2.3 mg/dl; and LDH, 392 IU/l. Creatinine clearance was 17 cc/min, and there was a total urinary protein of 838 mg/24 hr. Immunologic survey included a negative ANA, negative anti-DNA, negative RA, normal serum protein electrophoresis, and negative direct Coombs. The EKG showed low QRS voltage but was otherwise normal. The chest x-ray showed bilateral pleural effusions. An echocardiogram revealed 0.5-1 cm circumferential pericardial effusion. The pleural effusion was identified as a transudate with negative cultures; glucose, 113 mg/dl; negative cytology; WBC, 175/mm³; negative ANA and RA; and a low total complement of 33 µ/cc. The pericardial fluid findings were: LDH, 197 IU/l; total protein, 2.7 g/l; glucose, 265 mg/dl; WBC, 287/mm³, normal cytology; and negative cultures. A skin biopsy was consistent with scleroderma.

Because of the clinical features suggesting reduced cardiac output, a specific study was designed to determine whether the patient had sufficient pericardial effusion to explain his heart failure or had a restrictive cardiomyopathy. Right heart catheterization was performed and multiple pressures were obtained in the chambers of the right heart as well as in the wedge position (*Table I*). Using thermodilution technique with a narrow range between values, the average was 4.5 liters/minute. The pericardial space was aspirated; only 200 cc of fluid were obtained. Subsequent pressure studies showed no change in any of them. Specifically, there was a 10

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TABLE I

Right atrium	A Wave	12 mm Hg
	V Wave	10 mm Hg
	Mean	10 mm Hg
Right ventricle	Systolic	35
	End Diastolic	15
Pulmonary Artery	Systolic	35
	Diastolic	22
	Mean	28
Pulmonary Wedge	A Wave	23
	V Wave	23
	Mean	20
Cardiac Output		
Before pericardial aspiration		4.5 liter/minute
After pericardial aspiration		5.0 liter/minute

mm difference between the mean pressure in the right atrium and the wedge, consistent with restrictive cardiomyopathy rather than tamponade. Failure of the pressures to drop with the pericardial aspiration helped confirm the diagnosis of restrictive cardiomyopathy. There was a slight rise in cardiac output based upon removal of even this small amount of fluid. However, the added adrenergic effect of undergoing pericardiocentesis, although it was not traumatic, could have easily explained the slight rise in cardiac output following the procedure. Myocardial biopsy was obtained by the Stanford technique using the right internal jugular vein and revealed interstitial fibrosis and concentric narrowing of the arterioles consistent with the findings of scleroderma.

The patient developed progressive renal failure requiring hemodialysis within four weeks of his second admission.

Discussion

Progressive systemic sclerosis is generally a disease of insidious onset with fatigue, weight loss, diffuse stiffness and aching, skin changes, and Raynaud's phenomenon as early manifestations. Only rarely does the disease follow a rapidly progressive course with severe visceral involvement

over a period of six months.⁷ The incidence of cardiac involvement varies and is only rarely a presenting symptom. Clinical cardiac involvement was reported in only 26 of 309 patients reviewed by Medsger.⁷ McWhorter and Leroy⁸ discovered 15 patients with pericardial disease out of 210 patients with scleroderma studied retrospectively.

The reported incidence of clinical cardiac disease in scleroderma differs from the incidence found at autopsy. Bulkley reported that 26 of 52 patients had foci of myocardial fibrosis at autopsy.³ The use of echocardiography, however, has aided in the identification of cardiac disease in patients with scleroderma. Smith reported echocardiographic abnormalities in 37 of 54 patients (69%) with progressive systemic sclerosis and pericardial effusions in 22 (41%).⁹ This correlates well with autopsy findings.

The type of cardiac involvement has also been the subject of some controversy. Myocardial fibrosis, atrial or ventricular conduction disturbances,¹ and pericardial effusions have been felt to be most common. Fibrinous pericarditis was found in 17 of 52 patients by Bulkley³ and constrictive pericarditis was reported by Uhl.⁴

In this case the skin biopsy, done early in the clinical course, gave the initial clues as to the cause of the myocardial abnormality. The right ventricular pressure tracing had a "square root sign" which is characteristic of very limited ventricular distensibility associated with constrictive pericarditis, cardiac tamponade, or restrictive cardiomyopathy. However, it was only with aspiration of the scanty amount of pericardial fluid that we were able to confirm the absence of cardiac tamponade and the presence of a restrictive cardiomyopathy, both by abnormal tissue findings at myocardial biopsy, and by the failure of improvement of pressures with removal of the pericardial fluid.

Myocardial biopsy, well-described by Mason¹² and others, can be used safely even in patients with complicated medical problems with a risk less than standard heart catheterization. It can be of value in specific circumstances, as it was in this case, as it helped confirm the diagnosis of restrictive cardiomyopathy in progressive systemic sclerosis. This has rarely been reported.^{6, 10, 11}

References are available from Dr. Schurle, 400 West Fourth Street, McPherson KS 67460.

Intracranial Non-Hodgkin's Lymphoma

RAGHUNATH P. REDDI, M.D.* and DAVID N. NIEDEREE, M.D.,† *Wichita*

EXTRANODAL involvement is common in patients with non-Hodgkin's lymphoma (NHL), but isolated involvement of the central nervous system is estimated at only 1-6 per cent of the total, with 150 reported cases found in the world literature in 1975.¹ A recent summary of the relevant features² indicates that only the diffuse histiocytic lymphoma involves the parenchyma of the brain with clinical and radiologic features essentially the same as those of gliomas. Although the tumors are considered radiosensitive, the long term results of radiation therapy — with or without regional lymph node treatment — are disappointing with only six cases (4%) of the 150, disease-free for five or more years.

The role of chemotherapy has not been established. One case of recurrent primary NHL successfully treated with high-dosage methotrexate has been reported.³ Of 15 cases receiving prophylactic irradiation of the entire CNS without increase in the rate of local control, two also treated with cystine arabinoside showed no response to it.⁴

A case of extranodal NHL in the scalp with dural and brain involvement is presented.

Case Report

A 56-year-old American Indian female was admitted to St. Joseph Medical Center, Wichita, with the chief complaint of severe, throbbing, frontal headaches associated with nausea and vomiting during the previous month. A spinning sensation, with or without headaches was noted but without seizures, stroke, or epilepsy. For two days prior to admission she had been reported to be incoherent.

Physical examination disclosed the patient to be awake, alert, and oriented with normal vital signs. Two round elevations, each 4.0 cm in diameter, on the right parietal area of the skull and multiple small discrete lumps in the right frontal and temporal areas were noted. Cranial nerve and fundoscopic examinations were normal. The liver, spleen, and peripheral lymph nodes were not palpable.

Laboratory studies (CBC, urinalysis, SMAC 20) yielded normal results, and cytologic examination of

the cerebrospinal fluid (CSF) was negative for malignant lymphoma cells. Chest and KUB x-rays were normal as was bilateral mammography. Skull x-rays revealed increased osteoblastic and osteolytic change of the right parietal bone extending anteriorly into the frontal bone and inferiorly into the right temporal region with an associated soft tissue mass noted in the parietal area. CT scan (*Figure 1*) revealed a gross bony abnormality of the calvarium with intracranial extension and a soft tissue mass in the right parietal area. Arteriograms revealed an epidural mass with intracranial extension on the right causing a 1.0 cm shift of the midline structures to the left. Bone scan disclosed involvement of the cranial vault, especially the right frontal, parietal, and occipital bones, compatible with neoplasm. CT scans of the abdomen and pelvis and bone marrow examination showed no abnormality.

A right fronto-parietal craniotomy revealed a gelatinous, necrotic tumor involving the dura in the temporal and parietal regions. Biopsies were taken from the brain, bone, and dura.

Histopathology disclosed small lymphocytic-looking cells with hyperchromatic nuclei and nuclear membrane irregularities. Special stain showed reticulin fibers surrounding groups rather than individual cells and larger histiocytic-looking cells with

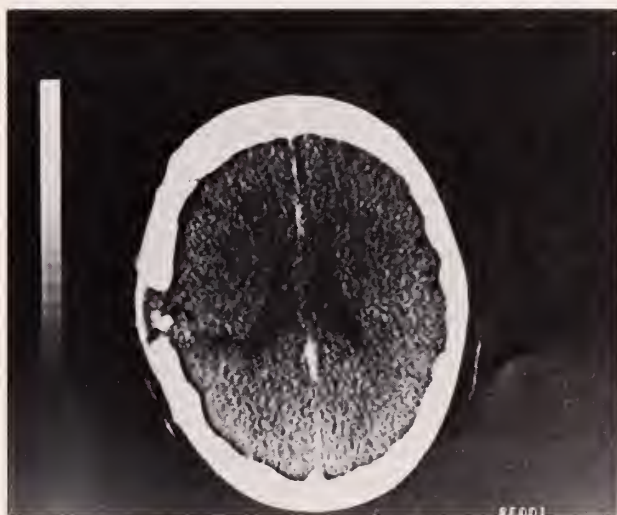


Figure 1. CT scan showing gross bony abnormality of the calvarium with intracranial extension and a soft tissue mass in the right parietal area.

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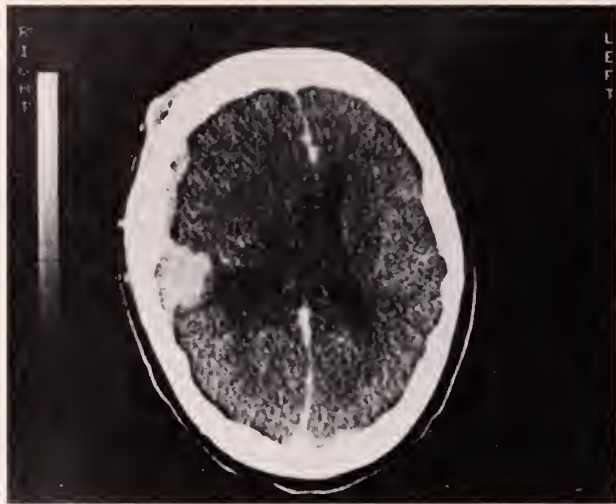


Figure 2. CT scan following radiation therapy.

sometimes foamy, sometimes nondescript, cytoplasm with granular brown pigment and, in most, PAS positive granules. Many of these cells were benign with multiple round nuclei but some had atypical nuclei. No Reed-Sternberg cells, frank osteoid formation, rosettes, whorling, or psammoma bodies were observed. Occasionally, the cells formed cuffs around small blood vessels in the brain tissue but not in the manner of a glioma. A diagnosis of poorly differentiated lymphocytic lymphoma was made.

Electron microscopy of the scalp lesion showed a malignant lymphoma of diffuse mixed small and large cell type (International Classification: diffuse, poorly differentiated lymphocytic lymphoma, Rapaport; mixed follicular center cell lymphoma, diffuse, Lukes).

Radiation therapy was instituted with a tumor dose of 4,500 rads (45 Gy) to the whole brain and 6,000 rads (60 Gy) to the right parietal and temporal areas delivered in 35 treatment days with a 4 MeV linear accelerator. After radiation a repeat scan gave no evidence of residual mass or enhancement in the right parietal-temporal area or midline shift and ventricular dilatation (Figure 2). There was some improvement in the hyperostosis in the calvarium and radiolucent areas of the right parietal bone.

A repeat CT scan six months later was within normal limits with more evidence of healing of the right calvarium. One year post diagnosis, the patient was doing well without evidence of recurrence.

Discussion

Intracranial non-Hodgkin's lymphoma is rare.^{1, 5, 6} This discussion examines the management of 92 cases (including the present one) as reported in ten articles. Pathologic titles include primary intracranial lymphosarcoma, primary reticulum cell sarcoma, reticuloendothelial neoplasm, primary malignant lymphoma of the brain, and extranodal non-Hodgkin's lymphoma.

Little change in therapy has occurred in the last 20 years. Radiosensitivity is apparent. Dosage of 4,000-5,000 rads to the brain with additional exposure of the tumor site to a total of 5,000-5,500 rads is necessary for longer survival;^{10, 11} 3,000-3,500 rads will have only a short term effect.¹² The need for whole brain irradiation has been demonstrated by the finding of multifocal disease at autopsy.⁷⁻¹¹ Unless cytological examination of the cerebrospinal fluid reveals malignant cells, irradiation of the cord has not been beneficial.^{2-4, 11-13}

Survival times in these reports range from less than a year to more than five years with the general observation that the larger dosages of irradiation as noted above are necessary for optimum effect. In some cases, the disease in the brain has been controlled, but death has ensued from disseminated disease.

Chemotherapy has, so far, not figured significantly in the treatment of this condition.^{14, 15} Its use has been reported only in conjunction with irradiation or after irradiation has failed. Consequently, its independent effect is uncertain. High dose methotrexate with leucovorin rescue has been reported,¹⁴ but irradiation combined with methotrexate raises the possibility of complications such as leucoencephalopathy. The use of adjuvant chemotherapy to eradicate occult disease is under study, and new approaches such as different fractionation schedules and the use of radiation sensitizers warrant study.

Summary

Primary non-Hodgkin's lymphoma of the central nervous system is rare with cases reported in the literature not exceeding 180. The case of primary NHL of the CNS presented here was treated successfully. The available literature has been reviewed and discussed in relation to radiation therapy, chemotherapy, and survivals.

References are available from Dr. Reddi, St. Joseph Medical Center, 3600 East Harry, Wichita KS 67218.



Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Diagnostic Approach to the Patient With Chronic Pancreatic Disease and Suspected Pancreatic Carcinoma

NORTON J. GREENBERGER, M.D.,* *Kansas City, Kansas*

CHRONIC ALCOHOLISM is a very significant health problem in the United States. Recent studies indicate that more than 90 per cent of Americans drink alcohol at some time in their lives, and some 30-40 per cent of young men have problems with alcohol. Of adults admitted to the hospital, approximately 10-15 per cent use alcohol in excess. It is highly likely that there is a considerable residue of patients with occult chronic pancreatitis secondary to use of excessive amounts of alcohol. It is not until more than 90 per cent of the exocrine pancreas is destroyed that patients develop obvious stigmata of chronic pancreatic disease such as diarrhea, weight loss, and other signs and symptoms suggestive of malabsorption.

Reviewed here is the diagnostic approach to the patient with chronic pancreatic disease with special emphasis on recognition of chronic pancreatitis secondary to chronic alcoholism. In addition, clues to the earlier diagnosis of pancreatic carcinoma and tests useful in establishing that diagnosis will be reviewed.

Etiology

Chronic inflammatory disease of the pancreas may take the form of relapsing chronic pancreatitis or chronic pancreatitis. As mentioned above, patients with chronic extensive destruction of the pancreas (*i.e.* less than 10% of exocrine function remaining) will usually demonstrate steatorrhea, azotorrhea, and other stigmata of malabsorption. The

causes of pancreatic exocrine insufficiency are listed in *Table I*. Alcoholism is the most common cause of clinically apparent pancreatic insufficiency in adults in the United States. However, in children and young adults, cystic fibrosis is the most frequent cause of exocrine pancreatic insufficiency. It should be emphasized that the diagnosis of pancreatic carcinoma must be excluded in any adult past age 50 years who presents with stigmata of pancreatic exocrine insufficiency.

Clinical Features and Diagnosis

Patients with chronic pancreatitis may present with symptoms similar to those found in acute pan-

TABLE I
CAUSES OF PANCREATIC EXOCRINE
INSUFFICIENCY

- Chronic pancreatitis
 - Associated with chronic alcoholism*
 - Associated with primary hyperparathyroidism
 - Hereditary pancreatitis
 - Hemochromatosis
 - Following traumatic pancreatitis
 - Etiologic factor(s) unknown
- Cystic fibrosis*
- Severe protein calorie malnutrition with hypoalbuminemia
- Following surgery
 - Subtotal gastrectomy (Billroth I, Billroth II anastomosis)
 - Truncal vagotomy and pyloroplasty
 - Whipple procedure
 - 95% pancreatectomy for chronic pancreatitis
- Neoplasms
 - Adenocarcinoma of the pancreas*
 - Islet cell carcinoma
 - Zollinger-Ellison Syndrome (gastrinoma)
 - Benign pancreatic tumors causing pancreatic ductal obstruction

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* Most common.

creatitis. However, pain may be continuous or intermittent. In a small percentage of patients (approximately 10%) pain may actually be absent. Characteristically, the pain is persistent, deep seated, often radiates to the back, and is unresponsive to various medications including antacids. It may be increased by ingestion of alcohol or large meals. Weight loss, abnormal stools, and other signs suggestive of malabsorption are prominent in pancreatitis. *Table II* lists clinical features that are useful in establishing a diagnosis of pancreatic insufficiency. First, the stool should be examined for undigested muscle fibers, as this is a highly specific test for maldigestion. Unfortunately, it is a relatively insensitive test and is only positive when more than 95 per cent of the exocrine pancreas has been destroyed. The presence of steatorrhea, diabetes, and pancreatic calcification should certainly suggest the diagnosis of chronic pancreatitis. However, this classical diagnostic triad is present in only 25 per cent of patients. Accordingly, additional tests to evaluate exocrine pancreatic function are often necessary.

At the present time, the most reliable tests are the secretin test alone or a combination of the secretin plus cholecystokinin-pancreozymin test. In this test the patient receives an intravenous injection of secretin (1 unit/kg), either alone or with cholecystokinin-pancreozymin, and duodenal and gastric secretions are collected separately. An abnormal secretin test is characterized by a sharply reduced volume of secretions (less than 2 ml/kg/hr), decreased peak bicarbonate concentration (less than 80

mEq/l), and decreased protein output. Approximately 40 per cent of patients with pancreatic insufficiency have impaired absorption of vitamin B₁₂. This abnormality is believed to be due to persistent and abnormal binding of vitamin B₁₂ to R proteins rather than intrinsic factor because the lack of pancreatic proteolytic enzymes which digest the R protein-B₁₂ complex. An important parameter which confirms the diagnosis of pancreatic exocrine insufficiency is a dramatic response to pancreatic enzyme replacement therapy. If a malabsorption syndrome is documented and if this is due to exocrine pancreatic insufficiency, then the institution of adequate doses of pancreatic enzyme replacement therapy should result in a gain in weight, amelioration of steatorrhea, and a marked decrease in stool nitrogen and excretion of meat fibers.

Cancer of the Pancreas

General Considerations

Carcinoma of the pancreas is now the fourth commonest cancer causing death in the United States; only cancer of the lung, colon, and breast occur more frequently. The incidence of pancreatic carcinoma has increased more than 300 per cent during the past 40 years. Men are affected 1.5 times as frequently as women. The incidence of pancreatic cancer is definitely increased in (1) patients with diabetes mellitus, (2) cigarette smokers, and (3) individuals exposed to certain industrial carcinogens such as benzidine and betanaphthylene. The presence of pancreatic calcification, the ingestion of a high fat diet, and ingestion of excessive amounts of coffee also have been linked to an increased risk of pancreatic cancer. However, the evidence in support of the latter three risk factors is controversial.

Clinical Features

The clinical features suggestive of pancreatic carcinoma are well known. Common symptoms include weight loss, abdominal pain, anorexia, indigestion, diarrhea suggestive of irritable bowel syndrome, persistent back pain, anxiety, and the new onset of depression and other psychiatric abnormalities. Pertinent physical findings include the presence of jaundice with obstructive features, hepatomegaly, palpable abdominal mass, palpable gallbladder (Courvoisier's sign), an abdominal bruit in the left upper quadrant due to compression of the splenic artery and vein by the tumor mass, ascites, edema, and migratory thrombophlebitis. However, in many cases, most of these features are not present and a patient may just have unexplained weight loss, abdominal pain, or back pain.

TABLE II
CLINICAL FEATURES AND DIAGNOSIS OF
PANCREATIC INSUFFICIENCY

- | | |
|---|--|
| ● Recurrent abdominal pain (~ 10% patients deny abdominal pain) | |
| ● Undigested muscle fibers in the stool (>5 per slide on microscopic examination) | |
| ● Steatorrhea* | } classical diagnostic triad
but present on only
25% of patients |
| ● Diabetes mellitus* | |
| ● Pancreatic calcification* | |
| ● Abnormal test of exocrine pancreatic function* | |
| Abnormal secretion or secretin + cholecystokinin pancreozymin test | |
| ↓ Volume secretin (< 2.0 ml/kg/hr) | |
| ↓ HCO ₃ concentration (< 80 mEq/l) | |
| ↓ Protein output | |
| ● Impaired absorption of vitamin B ₁₂ | |
| ● Response to pancreatic enzyme replacement therapy* | |
| ↑ weight | |
| ↓ steatorrhea | |
| ↓ stool nitrogen and excretion of meat fibers | |

* Most important

Clues to the earlier diagnosis of pancreatic carcinoma are summarized in *Table III*. Diagnosis of pancreatic carcinoma should be suspected in patients past the age of 50 years who present with any of the following findings:

- Sudden onset of diabetes without a predisposing cause such as obesity or positive family history.
- Onset of pancreatic exocrine insufficiency or an attack of acute pancreatitis after age 50 years without any predisposing cause.
- Unexplained weight loss greater than 10 per cent of normal body weight.
- Unexplained abdominal pain with no apparent explanation after a standard workup.
- Onset of persistent pain without obvious cause.

Useful Diagnostic Tests

Laboratory data are only occasionally helpful in

TABLE III
CLUES TO THE EARLIER DIAGNOSIS OF
PANCREATIC CARCINOMA

- Onset of diabetes mellitus after age 50 without any obvious predisposing factors (positive family history, obesity, drugs such as corticosteroids)
- Onset of pancreatic exocrine insufficiency or acute pancreatitis after age 50 without any obvious predisposing cause
- Weight loss of > 10% of normal body weight without obvious cause
- Onset of persistent abdominal pain with no apparent explanation after a standard workup
- Onset of persistent mid back pain with no apparent explanation after a standard workup

suggesting the diagnosis of pancreatic carcinoma. Serum amylase and lipase values are abnormal in only 10 per cent of cases and are not very helpful tests. Approximately 40 per cent of patients with pancreatic carcinoma are diagnosed as having diabetes mellitus or abnormal carbohydrate tolerance within the two year period antedating the diagnosis of pancreatic carcinoma. Anemia occurs in approximately one third of patients. However, frank pancreatic insufficiency occurs in approximately 10-15 per cent of patients. In patients who have developed jaundice, liver tests reveal a characteristically objective type pattern with a disproportionately increased serum alkaline phosphatase level, serum AST and ALT (SGOT and SGPT) values less than 300 units, normal serum proteins, and negative markers for B viral hepatitis.

The tests that are more helpful in the diagnosis of pancreatic carcinoma are listed in *Table IV*. Ultrasound studies have a sensitivity of approximately 70 per cent and specificity of 80 per cent. However, this test may be suboptimal in patients who are obese, have excess intraabdominal gas, ascites, or barium present from previous studies. Pancreatic function tests, such as the secretin test, cannot differentiate between carcinoma and chronic pancreatitis; they merely indicate that pancreatic function is abnormal. Endoscopic retrograde cholangiopancreatography (ERCP) is one of the more definitive means of establishing the diagnosis of pancreatic carcinoma. This test has a sensitivity of 85-90 per cent and a specificity of 90 per cent. Percutaneous transhepatic cholangiography (PTC) will often demonstrate the site of obstruction but cannot distinguish between a pancreatic or bile duct neoplasm as the cause of biliary

(Continued on page 64)

TABLE IV
TESTS USEFUL IN THE DIAGNOSIS OF PANCREATIC CARCINOMA

<i>Test</i>	<i>Sensitivity</i>	<i>Specificity</i>	<i>Comment</i>
Ultrasound	70%*	80%	Problems: Gas, obesity, ascites, barium
Pancreatic function tests	85%*	75%	Cannot differentiate between CA vs. chronic pancreatitis
ERCP (endoscopic retrograde cholangiopancreatography)	85-90%	90%	PTC (percutaneous transhepatic cholangiography) comparable
CT scan	60-90% (AV = 85%)	85%	More accurate than ultrasound; still misses small (< 2.0 cm) lesions
Angiography	65%	90%	
Percutaneous biopsy with ultrasound guidance	80-90%	90%	May obviate need for laparotomy

* Sensitivity of pancreatic disease.



Whatever it may produce, 1984 promises to be a year of competition. The *Angelenos* are whooping it up for the approaching Olympiad with an exuberance immoderate even by California standards. In the medical world, triplet acronyms are heralding a new day when competition will be the prime determinant of patient care. And on the political front, the quadrennial ritual of picking candidates for the presidency, and then the victim, will have heads ringing long before November. True, the Republicans will have a relatively quiet time of it — even, so it is reported at this point, to reducing their convention sessions from the planned four days to three since the results are a foregone conclusion. The Democrats, however, can be counted on to intensify their intramural competition, and after the summer ceremonies, both sides (and numerous lesser aspirants) will engage in the traditional hyperbolic shoot-out.

It seems appropriate, then, to consider one of the more recent phenomena on the political scene, the political action committee (or PAC, which explanation must surely be a waste of space).

Probably, apart from certain religious instructions, no admonition has been voiced so repeatedly by certain interested parties as the advice to individuals to become personally involved in the political process. The repetition is, in itself, an indication that said individuals have either failed to respond or have done so in such a fragmentary way that they have derived nothing more than a sense of futility — and politicians have given them little heed.

Organizations commanding ample finances have never lacked for access to the political ear and, taking a cue from this, labor mobilized its formidable financial resources and, as a means of applying them, devised the term, political action committee, a triumph in euphemism. The prompt success of the effort was evident in the grousing of other organizations (including medicine) over such maneuverings which abated only as they, one by one, emulated the process. The PACs gave their organizations the means to assure being heard (read “clout”), at least regarding their own interests. Individual members were able to feel some sense of contact with the process, as well as some relief from the nagging of the political activists, with relatively little financial

pain. The politicians were grateful since, by making periodic expressions of approval of their PAC contributors, they were assured of more consistent transfusions than when relying on the uncertain affections of individuals.

There have been problems, of course. The PACs were obviously locked into their organizational commitments and therefore suspect in the eyes of outsiders, particularly those not aligned with a PAC-sponsoring group. Moreover, the PACs had to reconcile, when they finally got around to any specific endorsement, the widely varying political tempers within their ranks since the component individuals have by no means been unanimous in their approval of their own PACs’ efforts. And legal restrictions have required a degree of separation of the PACs from their sponsoring organizations, a separation which, whatever the effect on financial purity, has allowed some individuals to drop through and has also weakened their claim of speaking for the whole group.

But the fact is that the PACs may have been too successful. In recent years, they have attracted increasing attention from various observers who, whether unable to identify with a specific PAC or disgruntled by their performance, have mounted vigorous objections to their power. They see the evils of self-service rather than the virtues of enlightened political participation. Even as the PAC philosophy claims promotion of a broader political base with greater effect for organizational purposes (and by extension for the public good), its critics interpret it as the corruptive pressure of special interests. As ever, political activity promotes parochial criticism and the age-old contentions are played out against a different backdrop.

So the political year is moving forward, and whatever the merits of the objections, they should provide a desirable stimulus to the PACs for reassessment of their policies, methods, and objectives. The intensity of the political activity this year will certainly provide them with a suitable climate since both the virtues and the vices will be brought into relief. This, in turn, will demonstrate whether they have more to offer than any other system of incorporating the politically innocent or reluctant into what is a vital public process or are simply instruments of power belying their civic aspirations. Nothing succeeds like success, but success in itself exposes directions for change if it is to be sustained — as, we hope, 1984’s numerous competitors know. It should be an interesting year. — *D.E.G.*

AMA House of Delegates

Highlights of 1983 Interim Meeting, Los Angeles

JCAH

Joint Commission on Accreditation of Hospitals (JCAH) medical staff provisions was a dominant topic. Two broad issues considered were: (1) Physician responsibility for patients admitted to hospitals by limited licensed practitioners, and (2) The ability of the individual hospital and medical staff to determine which categories of limited licensed practitioners may be considered for medical staff membership.

Following lengthy debate, the House adopted the following policy statement:

- That it be the policy of the American Medical Association that the hospital medical staff may grant admitting privileges to appropriately credentialed limited licensed practitioners in accordance with state law and *in accordance with the criteria for standards of medical care established by the individual hospital medical staff.*
- That it be the policy of the American Medical Association that hospital admitting privileges be granted in accordance with state law and in accordance with criteria for standards of medical care established by the individual hospital medical staff.

Indemnity vs UCR

The House reaffirmed Association policy supporting:

- Freedom for physicians to choose the method of payment for their services and to establish fair and equitable fees.

- Freedom of patients to select their source of care.
- Neutral public policy and fair market competition among alternative health care delivery and financing systems.

The matter of Indemnity vs UCR as the basis for physician reimbursement will be further studied and a report presented at the AMA annual meeting in June 1984.

DRG Based Prospective Payment

The House filed a status report on regulations implementing the Prospective Payment System for Hospitals. In a related action, the House voted to:

- Endorse the concept that any system for reimbursement for physicians' services be independent of reimbursement systems for other providers of health care.
- Continue to oppose expansion of prospective payment systems until such time as they have been adequately evaluated with respect to their impact on the quality, cost, and access to medical care.

Surrogate Mothers

The House approved a Judicial Council report that concludes:

The Judicial Council believes that surrogate motherhood presents many ethical, legal, psychological, societal, and financial concerns and does not represent a satisfactory reproductive alternative for people who wish to become parents.

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Not after: physicians and hospitals are required to collect sales taxes from their patients.

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March 15, 1984

and

Jes Olesen, M.D., Hellerup, Denmark

at

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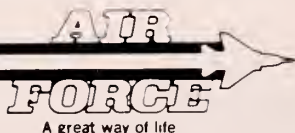
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BRIEF SUMMARY

PROCARDIA® (nifedipine) CAPSULES

For Oral Use

INDICATIONS AND USAGE: I. **Vasospastic Angina:** PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation; 2) angina or coronary artery spasm provoked by ergonovine; or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

II. **Chronic Stable Angina (Classical Effort-Associated Angina):** PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS:

Known hypersensitivity reaction to PROCARDIA.
WARNINGS: Excessive Hypotension: Although in most patients the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers, if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate the peripheral edema from the effects of increasing left ventricular dysfunction.

Drug interactions: Beta-adrenergic blocking agents. (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates. PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis. Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility. When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy. Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients; transient hypotension in about 5%; palpitation in about 2%; and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT, and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069-2600-66), 300 (NDC 0069-2600-72), and unit dose (10x10) (NDC 0069-2600-41). The capsules should be protected from light and moisture and stored at controlled room temperature 59° to 77° F (15° to 25° C) in the manufacturer's original container.

More detailed professional information available on request.

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Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%).



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* Procardia is indicated for the management of:

- 1) Confirmed vasospastic angina
- 2) Angina where the clinical presentation suggests a possible vasospastic component.
- 3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

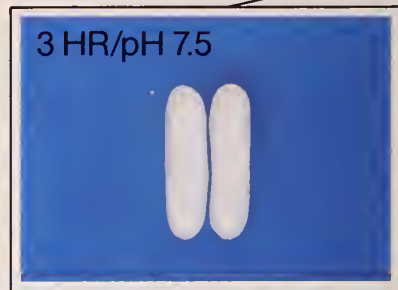
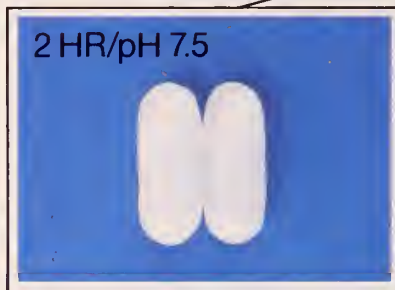
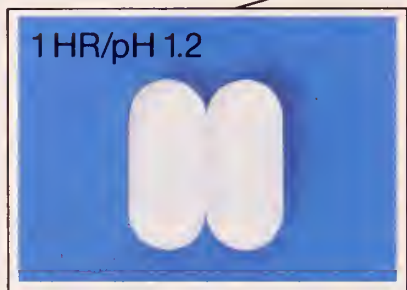
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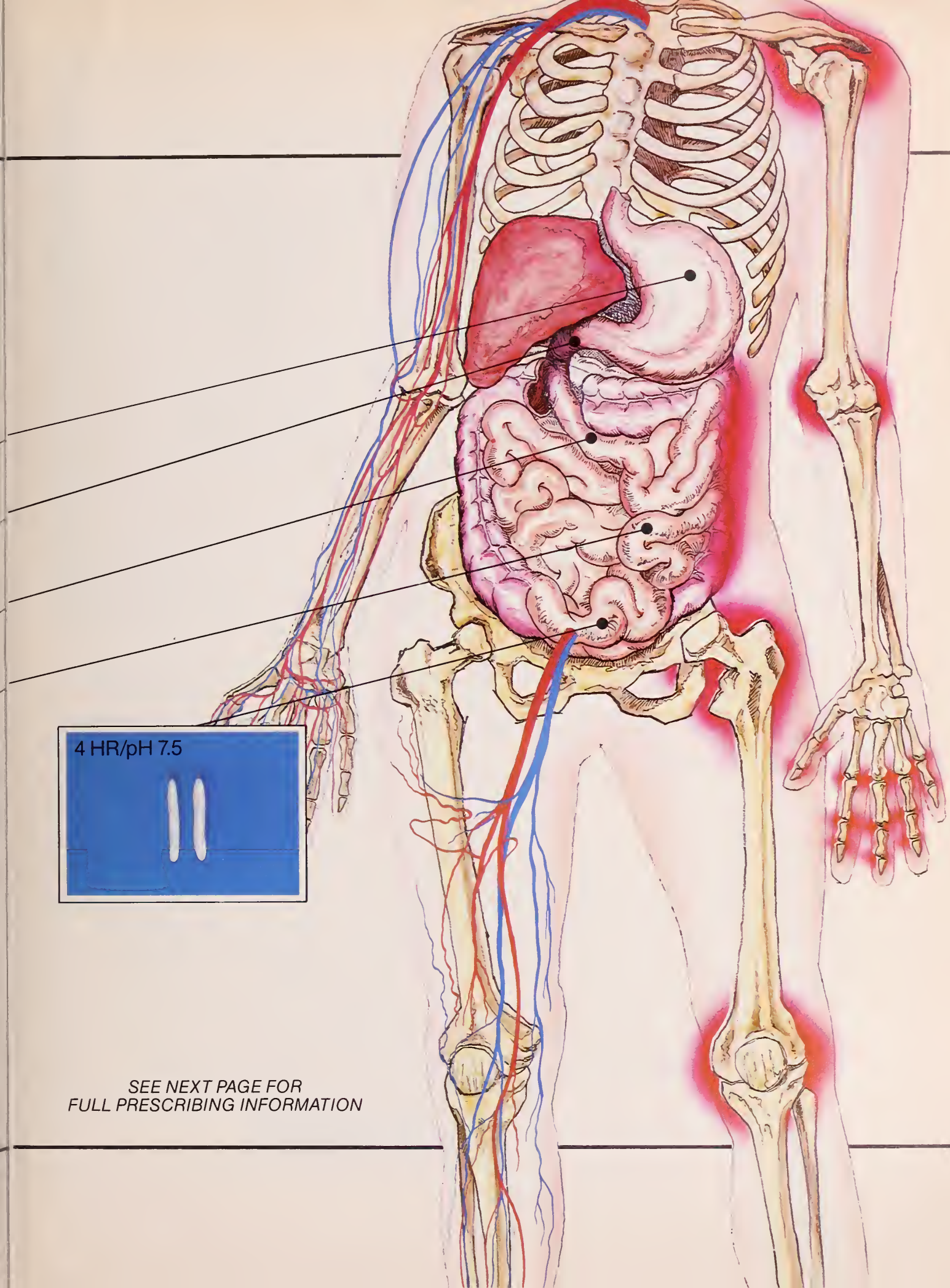
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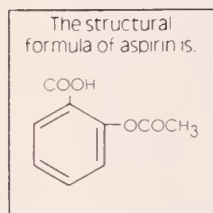


4 HR/pH 7.5

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ZORprin (ASPIRIN) Zero-Order Release

DESCRIPTION: Each capsule-shaped tablet of Zorprin contains 800 mg of aspirin, formulated in a special matrix to control the release of aspirin after ingestion. The controlled availability of aspirin provided by Zorprin approximates zero-order release, the *in vitro* release of aspirin from the tablet matrix is linear and independent of the concentration of the drug. **CLINICAL PHARMACOLOGY:** Aspirin, as contained in Zorprin, is a salicylate that has demonstrated anti-inflammatory and analgesic activity. Its mode of action as an anti-inflammatory and analgesic agent may be due to the inhibition of synthesis of prostaglandins, although its exact mode of action is not known. \square Zorprin dissolution is pH-dependent. *In vitro* studies have shown very little aspirin to be released in acidic solutions, whereas, Zorprin releases the majority of its aspirin (90%) in a zero-order mode at a neutral to alkaline pH. It is this pH dependence of Zorprin that reduces direct contact between aspirin and the gastric mucosa, resulting in a reduction of its gastrointestinal side-effect potential. \square Bioavailability data for Zorprin have confirmed that plasma levels of salicylic acid and acetylsalicylic acid can be measured 24 hours after a single oral dose. This substantiates a twice daily dose regimen. Multiple dose bioavailability studies showed similar steady-state salicylate levels for Zorprin as for conventional release aspirin using the same total daily dose. Long-term monitoring of salicylate levels showed no signs of accumulation once steady-state levels were reached (4-6 days). \square Studies of *in vivo* prostaglandin levels (PGE₂) have shown Zorprin plasma levels of salicylic acid and acetylsalicylic acid to reduce PGE₂ levels 14 hours after a single oral 800 mg dose while an equivalent dose of aspirin produced a reduction of PGE₂ levels only through six hours. Zorprin's effect on prostaglandins other than PGE₂ has not been determined. \square Salicylates are excreted mainly by the kidney, and from studies in humans it appears that salicylate is excreted in the urine as free salicylic acid (10%); salicylic acid (75%); salicylic phenolic (10%); acyl glucuronides (5%) and gentisic acid (<1%). **INDICATIONS & USAGE:** Zorprin is indicated for the treatment of rheumatoid arthritis and osteoarthritis. The safety and efficacy of Zorprin have



not been established in those rheumatoid arthritis patients who are designated by the American Rheumatism Association as Functional Class IV (incapacitated, largely or wholly bedridden, or confined to wheelchair, little or no self-care). \square In patients treated with Zorprin for rheumatoid arthritis and osteoarthritis, the anti-inflammatory action of Zorprin has been shown by reduction in pain, morning stiffness and disease activity as assessed by both the investigators and patients. \square In clinical studies in patients with rheumatoid arthritis and osteoarthritis, Zorprin has been shown to be comparable to conventional release aspirin in controlling the aforementioned signs and symptoms of disease activity and to be associated with a statistically significant reduction in the milder gastrointestinal side effects (see ADVERSE REACTIONS). Zorprin may be well tolerated in some patients who have had gastrointestinal side effects with conventional release aspirin, but these patients when treated with Zorprin should be carefully followed for signs and symptoms of gastrointestinal bleeding and ulceration. \square Since there have been no controlled trials to demonstrate whether or not there is any beneficial effect or harmful interaction with the use of Zorprin in conjunction with other nonsteroidal anti-inflammatory agents (NSAIs), the combination cannot be recommended (see Drug Interactions). **Because of its relatively long onset of action, Zorprin is not recommended for antipyresis or for short-term analgesia.** **CONTRAINDICATIONS:** Zorprin should not be used in patients known to be hypersensitive to salicylates or in individuals with the syndrome of nasal polyps, angioedema, bronchospastic reactivity to aspirin, renal or hepatic insufficiency, hypoprothrombinemia or other bleeding disorders. Zorprin is not recommended for children under 12 years of age, it is contraindicated in all children with fever accompanied by dehydration. **WARNINGS:** Zorprin should be used with caution when anticoagulants are prescribed concurrently, since aspirin may depress platelet aggregation and increase bleeding time. Large doses of salicylates may have hypoglycemic action and enhance the effect of the oral hypoglycemics, concomitant use therefore is not recommended. However, if such use is necessary, dosage of the hypoglycemic agent must be reduced. The hypoglycemic action of the salicylates may also necessitate adjustment of the insulin requirements of diabetics. \square While salicylates in large doses have a uricosuric effect, smaller amounts may reduce water excretion and increase serum uric acid. \square **USE IN PREGNANCY:** Aspirin can harm the fetus when administered to pregnant women. Aspirin interferes with maternal and infant hemostasis and may lengthen the duration of pregnancy and parturition. Aspirin has produced teratogenic effects and increases the incidence of stillbirths and neonatal deaths in animals. \square If this drug is used during pregnancy, or if the patient becomes pregnant while taking this drug, the patient should be apprised of the potential hazard to the fetus. \square Aspirin should not be taken during the last 3 months of pregnancy. **PRECAUTIONS:** Appropriate precautions should be taken in prescribing Zorprin for patients who are known to be sensitive to aspirin or salicylates. Particular care should be used when prescribing this medication for patients with erosive gastritis, peptic ulcer, mild diabetes or gout. As with all salicylate drugs, caution should be exercised in prescribing Zorprin for those patients with bleeding tendencies or those on anticoagulants. \square In order to avoid exacerbation of disease or adrenal insufficiency, patients who have been on prolonged corticosteroid therapy should have their therapy tapered slowly rather than discontinued abruptly when Zorprin is made a part of the treatment program. \square Patients receiving large doses of aspirin and/or prolonged therapy may develop mild salicylate intoxication (salicylism) that may be reversed by dosage reduction. \square Salicylates can produce changes in thyroid function tests. \square Salicylates should be used with caution in patients with severe hepatic damage, preexisting hypoprothrombinemia, Vitamin K deficiency and in those undergoing surgery. \square Since aspirin release from Zorprin is pH dependent, it may change in those conditions where the gastric pH has been increased as a result of antacids, gastric secretion inhibitors or surgical procedures. **Drug Interactions:** (See **WARNINGS**) Aspirin may interfere with some anticoagulant and antidiabetic drugs. Drugs which lower serum uric acid by increasing uric acid excretion (uricosurics) may be antagonized by the concomitant use of aspirin, particularly in doses less than 2.0 grams/day. Nonsteroidal anti-inflammatory drugs may be competitively displaced from their albumin binding sites by aspirin. This effect may negate the clinical efficacy of both drugs. Also, the gastrointestinal inflammatory potential of nonsteroidal anti-inflammatory drugs may be potentiated by aspirin. The combination of alcohol and aspirin may increase the risk of gastrointestinal bleeding. \square Aspirin may enhance the activity of methotrexate and increase its toxicity. \square Sodium excretion produced by spironolactone may be decreased in the presence of salicylates. Concomitant administration of other anti-inflammatory drugs may increase the risk of gastrointestinal ulceration. Urinary alkalinizers decrease aspirin's effectiveness by increasing the rate of salicylate renal excretion. Phenobarbital decreases aspirin's effectiveness by enzyme induction. **Pregnancy Category D.** See **WARNINGS** Section. **Nursing Mothers:** Salicylates have been detected in the breast milk of nursing mothers. Because of the potential for serious adverse reactions from aspirin in nursing infants, a decision should be made whether to discontinue nursing or discontinue the drug, taking into account the benefits of the drug to the mother. **ADVERSE REACTIONS: Hematologic:** Aspirin interferes with hemostasis. Patients with a history of blood coagulation defects or receiving anti-coagulant drugs or with severe anemia should avoid Zorprin. Aspirin used chronically may cause a persistent iron deficiency anemia. **Gastrointestinal:** Aspirin may potentiate peptic ulcer, and cause stomach distress or heartburn. Aspirin can cause an increase in occult bleeding and in some patients massive gastrointestinal bleeding. However, the greatest release of active drug from Zorprin is designed to occur in the small intestine over a period of time. This has resulted in fewer symptomatic gastrointestinal side effects. **Allergic:** Allergic and anaphylactic reactions have been noted when hypersensitive individuals have taken aspirin. Fatal anaphylactic shock, while not common, has been reported. **Respiratory:** Aspirin intolerance, manifested by exacerbations of bronchospasm and rhinitis, may occur in patients with a history of nasal polyps, asthma, or rhinitis. The mechanism of this intolerance is unknown but may be the result of aspirin-induced shunting of prostaglandin synthesis to the lipoxigenase pathway and the liberation of leukotrienes, e.g. slow-reacting substance of anaphylaxis. **Dermatologic:** Hives, rashes, and angioedema may occur, especially in patients suffering from chronic urticaria. **Central Nervous System:** Taken in overdoses, aspirin provides stimulation which may be manifested by tinnitus. Following initial stimulation, depression of the central nervous system may be noted. **Renal:** Aspirin rarely may aggravate chronic kidney disease. **Hepatic:** High doses of aspirin have been reported to produce reversible hepatic dysfunction. **OVERDOSAGE:** Overdosage, if it occurs, would produce the usual symptoms of salicylism: tinnitus, vertigo, headache, confusion, drowsiness, sweating, hyperventilation, vomiting or diarrhea. Plasma salicylate levels in adults may range from 50 to 80 mg/dl in the mildly intoxicated patient to 110 to 160* mg/dl in the severely intoxicated patient. An arterial blood pH of 7.1 may indicate serious poisoning. The clearance of salicylates in children is much slower than adults and should receive due consideration when aspirin overdoses occur in infants, salicylate half-lives of 30 hours have been reported in infants 4-8 months old. Treatment for mild intoxication should include emptying the stomach with an emetic, or gastric lavage with 5% sodium bicarbonate. Individuals suffering from severe intoxication should, in addition, have forced diuresis by intravenous infusions of sodium bicarbonate and dextrose or sodium lactate. In extreme cases, hemodialysis or peritoneal dialysis may be required. \square (*A plasma salicylate level of 160 mg/dl in an adult is usually considered lethal.) **DOSEAGE & ADMINISTRATION:** In order to achieve a zero-order release, the tablets of Zorprin should be swallowed intact. \square Breaking the tablets or disrupting the structure will alter the release profile of the drug. \square It is recommended that Zorprin be taken with sufficient quantities of fluids (8 oz. or more). **Adult Dosage:** For mild to moderate pain associated with rheumatoid arthritis and osteoarthritis, the recommended initial dose of Zorprin is 1600 mg (2-800 mg tablets) twice a day. Because of Zorprin's prolonged release of aspirin into the bloodstream, Zorprin tablets may be taken as a b.i.d. dose. Further adjustment of the dosage should be determined by the physician, based upon the patient's response and needs. Since it will take 4-6 days to reach steady-state levels of salicylic acid with Zorprin, it is recommended dosages be given for at least one week before further adjustment. In general, patients with rheumatoid arthritis seem to require higher doses of Zorprin than do patients with osteoarthritis. **Zorprin is not recommended for children below the age of 12.** **HOW SUPPLIED:** Zorprin Tablets 800 mg; plain, white capsule-shaped tablets. \square Bottles of 100 Tablets — NDC 0524-0057-01 \square **Caution:** Federal law prohibits dispensing without prescription. \square U.S. Patent No. 4,308,251 \square **Manufactured and Distributed by: BOOTS PHARMACEUTICALS, INC., Shreveport, Louisiana 71106 U.S.A.**

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Current Comment

(Continued from page 55)

tract obstruction. CT scans have an average sensitivity of approximately 85 per cent and reasonably good specificity. CT scanning appears to be more accurate than ultrasound. However, lesions smaller than 2.0 cm are still frequently missed. Selective and superselective angiography has a somewhat diminished sensitivity (65%) but good specificity. Recently, several studies have indicated that percutaneous biopsy with ultrasound guidance has a high sensitivity and specificity for the diagnosis of pancreatic carcinoma. Importantly, a positive biopsy in this manner may obviate the need for exploratory laparotomy.

Summary

With a considerable segment of the United States population ingesting excessive amounts of alcohol, it is highly likely that there are substantial numbers of patients with unrecognized chronic pancreatic dis-

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ease. The commonest cause of chronic pancreatitis in adults in the U.S. is chronic alcoholism. Cystic fibrosis is the commonest cause of pancreatic insufficiency in children and adolescents. Diagnosis of chronic pancreatitis should be suspected in all chronic alcoholic patients who present with diarrhea, weight loss, or findings suggestive of malabsorption. The presence of steatorrhea, pancreatic calcification, and diabetes — *i.e.*, the classic diagnostic triad — establishes the diagnosis of chronic pancreatitis and pancreatic exocrine insufficiency. However, this classical diagnostic triad is present in only one fourth of the patients. Accordingly, some other test of pancreatic function needs to be carried out in this setting. In any adult past the age of 50 years who presents with stigmata of pancreatic exocrine insufficiency, the diagnosis of pancreatic carcinoma must be excluded. Clues to the earlier diagnosis of pancreatic carcinoma and tests useful in this regard are summarized above.

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Contraindications: Known hypersensitivity to flurazepam HCl; pregnancy. Benzodiazepines may cause fetal damage when administered during pregnancy. Several studies suggest an increased risk of congenital malformations associated with benzodiazepine use during the first trimester. Warn patients of the potential risks to the fetus should the possibility of becoming pregnant exist while receiving flurazepam. Instruct patient to discontinue drug prior to becoming pregnant. Consider the possibility of pregnancy prior to instituting therapy.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, e.g., excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. **Adults:** 30 mg usual dosage; 15 mg may suffice in some patients. **Elderly or debilitated patients:** 15 mg recommended initially until response is determined.

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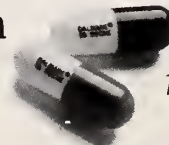
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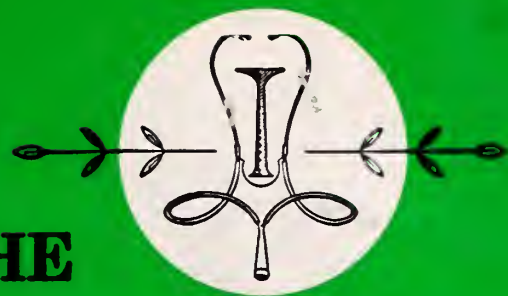
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The JOURNAL of the KANSAS MEDICAL SOCIETY

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Nominating Committee Report

The KMS Nominating Committee has submitted the following slate of candidates for offices for the election to be held at the annual meeting May 3-6, 1984, in Hutchinson:

PRESIDENT: F. Calvin Bigler, M.D., Garden City

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Richard Meidinger, M.D., Topeka; and Roger D. Warren, M.D., Hanover

SPEAKER: G. Rex Stone, M.D., Manhattan

VICE SPEAKER: Herbert Fransen, M.D., Newton; and Edwin D. Rathbun, M.D., Liberal

AMA DELEGATE: Alex Scott, M.D., Junction City

AMA ALTERNATE: Warren E. Meyer, M.D., Wichita

AMA DELEGATE: Kermit G. Wedel, M.D., Minneapolis

AMA ALTERNATE: Linda D. Warren, M.D., Hanover

The KMS Nominating Committee encourages additional nominations, which may be presented by delegates at the annual meeting.

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1:00 Keynote: DRGs IN HEALTH CARE

1:45 THE OTHER VIEW: THE NON-PARTICIPATING PHYSICIAN

2:15 HEALTH CHANGES AND MARKETING

3:00 Break

3:10 MEDICAL RECORDS UNDER PROSPECTIVE PAYMENT SYSTEM

4:00 CURRENT ISSUES IN MEDICAID REIMBURSEMENT

4:40 Wrapup, Evaluations, CEU Test (as may be applicable)

5:00 Adjournment

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Marlon Dauner, Sr. Vice President External Affairs, Blue Cross and Blue Shield of Kansas
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Drugs should be referred to by generic names; trade names may follow in parentheses if useful. All **units of measure** must be given in the metric system.

THE JOURNAL does not publish **references**. However, a reference list should be submitted with the manuscript and superscripts placed in the text in sequential order. Readers will be referred to the author(s) for references.

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References:

1. Stone PH, Turin ZG, Muller JE. Efficacy of nifedipine therapy for refractory angina pectoris. *Am Heart J* 104 672-681, September 1982
2. Antman E, Muller J, Goldberg S, et al. Nifedipine therapy for coronary-artery spasm: Experience in 127 patients. *N Engl J Med* 302 1269-1273, June 5, 1980

BRIEF SUMMARY

PROCARDIA® (nifedipine) CAPSULES

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INDICATIONS AND USAGE: I. **Vasospastic Angina:** PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation; 2) angina or coronary artery spasm provoked by ergonovine; or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm, or when angina is refractory to nitrates and/or adequate doses of beta blockers.

II. **Chronic Stable Angina (Classical Effort-Associated Angina):** PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients, the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General. Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug interactions: Beta-adrenergic blocking agents. (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates. PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy: Category C. Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients; transient hypotension in about 5%; palpitation in about 2%; and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LOH, SGOT and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

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**Thirty-Eighth
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University of Kansas
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The Vice Chancellor's Letter — 1984

The University of Kansas School of Medicine-Kansas City

D. KAY CLAWSON, M.D.,* *Kansas City, Kansas*

IN MY TRAVELS around the state, speaking and listening to physicians in many different settings, I have heard several recurrent themes, some very relevant to the University of Kansas School of Medicine. The theme heard most often involves the supply of health professionals. Space does not permit me to elaborate upon how, in such a relatively short period of time, what was widely viewed as a state of national deprivation could have been transformed into what some describe as a state of near satiety — the well publicized issue of the supply and distribution of health professionals. Immediate needs and gloomy forecasts in the 1950s prompted an array of public policy decisions in the 1960s and beyond, decisions that addressed the issue at national, state, and local levels. The effects on medical education are familiar to most readers of the *Journal*: more medical schools, more students, more residents, greater emphasis upon primary care, incentives to influence choice of specialty and location of practice, etc. Analogous measures were directed to other sectors of the health professions.

The outcome of these initiatives has been clearly evident on a national scale. The supply and distribution of health professionals has improved everywhere, Kansas included. True — we are not yet out of the woods. Areas of our state, especially in western Kansas, continue to suffer from a shortage of primary care physicians. Moreover, the rate of increase in our supply of primary care physicians — especially those in family medicine — has not matched that of the other specialties. The Kansas Department of Health and Environment forecasts a continuing, albeit reduced, shortage of family physicians into the 1990s.

Overall, however, thanks in large measure to the Kansas Medical Scholarship program, prospects for continuing improvement are bright. It is important, therefore, that the School of Medicine re-evaluate the size of its entering class and, if necessary, modify it to arrive at the most appropriate match between need, demand, and production. This applies to *all* of the health professions schools at the College of

Health Sciences. We should not exclude qualified and motivated residents of Kansas who apply for admission to UKSM. On the other hand, we should not lower our admission standards merely to fill out a quota of 200 entering students. Applications to medical school have decreased considerably, in Kansas and nationally. In the face of that reality, and for the sake of society and our profession, we will ensure a continuing high quality of medical care only by admitting high quality students into medical school, even if the number is below our admissions ceiling.

Because of these shifting priorities, we can now redirect our energies to the process of educating health professionals and to the quality of the programs that nourish that process. We are obliged to continuously assess the quality of preparation of students for their future as health professionals. Our curriculum must emphasize problem-solving skills as well as medical craftsmanship. We must fully exploit our potential in biomedical research — the royal road to excellence in patient care and in education. We must show our students the essential parallel between problem solving in clinical medicine and problem solving as the driving force behind scientific research. We must strive to develop our areas of strength into high quality research and patient care that involve our students, residents, and faculty. We must redirect our existing resources to develop our potential for excellence and thereby attract new resources for further growth.

Excellence should be the standard by which we measure all of our efforts. Demonstrable quality and high achievement: there is *no* substitute for excellence when it comes to caring for the ill and to preparing young people to meet that task. If we, at your school of medicine, cannot afford to be excellent, we cannot afford to conduct the program at all.

I invite you to join me in meeting the exciting challenges ahead. We share a common interest. For my part, I will do all that I can to articulate and strengthen the bonds that unite us in a common destiny. We should “Look to the past only for the lessons we learn; live for today for the sheer joy of being alive; plan for the future to insure that what *should* be *will* be.”

* Executive Vice Chancellor, UKSM Kansas City and Wichita.



Diagnostic Cholescintigraphy

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ACUTE CHOLECYSTITIS is a common diagnostic consideration in cases of abdominal pain. Early identification of those patients with acute cholecystitis permits early surgical management without additional mortality or complication.¹⁻³ Between 16 and 20 per cent of those patients initially diagnosed as having acute cholecystitis are finally determined to have other causes for their symptoms and may undergo unnecessary exploratory laparotomy.⁴ Severe cholecystitis may be present in the elderly without abdominal tenderness or febrile response.^{5, 6} A rapid, noninvasive, sensitive, and specific test to identify the altered physiology of acute cholecystitis would be useful.

Cholescintigraphy has developed into such a test. Experience with cholescintigraphy at the University of Kansas Medical Center is the basis of this report.

Method

Between January 1, 1979 and October 31, 1982, 90 patients underwent cholescintigraphy. After a period of 3-21 months followup, chart review ascertained the clinical reason for the study, the result of the study, and the clinical outcome of each patient.

Scintigraphic examinations were performed 2-24 hours after the previous meal. Three millicuries of paraisopropyl iminodiacetic acid (PIPIDA, Medi-Physics, Inc., Emoryville, California) was injected intravenously. Images were obtained on a Searle large field of view gamma camera or on a standard field of view Ohio Nuclear 415 gamma camera. With each camera, a low energy all purpose collima-

tor was used. Twenty 30-second images were obtained for the first ten minutes in the anterior view followed by an additional image every ten minutes for an hour. When structures were seen that might be the gallbladder, right and left anterior oblique and right lateral views were obtained to differentiate between anteriorly located gallbladder and underlying posteriorly located common duct and duodenum. Occasionally images were obtained at four hours after injection when there was delayed visualization or nonvisualization of the gallbladder or small bowel. Each static image contained 500,000 counts. The initial dynamic images were judged as to the adequacy of hepatocellular function based on the rapidity of blood clearance and liver uptake of PIPIDA. The scintigraphic diagnosis of acute cholecystitis was considered present when there was a failure to visualize the gallbladder by four hours, indicating cystic duct obstruction.

The actual presence of acute cholecystitis was determined by histological examination of surgical tissue specimen when possible and the medical diagnosis of acute cholecystitis was accepted after extensive medical followup.

Results

Of the 90 patients reviewed, 62 scans were performed on patients with abdominal pain in whom acute cholecystitis was a diagnostic consideration. Seventeen examinations were performed on patients with prior cholecystectomy to investigate the possibility of bile leaks or biliary obstruction. In ten patients, the clinical question was one of biliary atresia or hepatocellular dysfunction.

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Noninvasive Measurement of O₂/CO₂

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REFINEMENT of rapid analytic methods for determining oxygen and carbon dioxide tensions has revolutionized care of patients with respiratory failure. Until recently the prime indices used for assessment of gas exchange have been the tensions of oxygen and carbon dioxide in arterial blood (PaO₂ and PaCO₂), which require either serial arterial punctures or an indwelling arterial cannula. Although complications resulting from arterial puncture are infrequent, there are compelling reasons to develop noninvasive methods for measuring gas. Effective monitoring of vital functions depends as much on continuity as on reliability. Profound changes in respiratory function can occur between serial arterial determinations. Although continuous readouts are obtainable via an arterial line, their accuracy depends on sustained performance of the indwelling sensor, the weakest link in the system. External sensors, once their individual peculiarities are recognized, are more easily adapted to continuous recording devices, thus providing the clinician with unremitting surveillance of vital functions. In this review, several currently available noninvasive methods of gas measurement will be briefly evaluated. These include end-tidal carbon dioxide monitoring (ETCO₂), ear oximetry and pulse oximetry, which measure hemoglobin saturation (SaO₂), and transcutaneous oxygen and carbon dioxide determinations (P_{tc}O₂ and P_{tc}CO₂).

End-Tidal Carbon Dioxide Analyzers

Continuous breath-by-breath analysis of expired air can be monitored by two different types of instruments: the infrared monitor and the mass spectrometer. The ETCO₂, the CO₂ concentration (or partial pressure) measured at end-expiration, is accurately determined by either device. Theoretically, the ETCO₂ should approximate the value of the mixed venous PCO₂. However, the ETCO₂ is subject to many variations, depending on the rate and amplitude of ventilation, matching of pulmonary ventilation and perfusion, changes in circulatory transport

of CO₂, minute volume production of CO₂, and the amount of rebreathing at the site of the monitoring sensor. There can be an arterial to end-tidal gradient of -6 to +20 torr.¹ Patients with abnormal lungs almost always have some ventilation-perfusion abnormalities, resulting in dilution of expired CO₂ by air from alveoli in which no gas exchange has occurred. The ETCO₂ can thus be much lower than the corresponding blood CO₂ tensions. An extreme example of this is often seen immediately following pulmonary embolism. On the other hand, rebreathing due to any cause will raise the ETCO₂, and may reverse the alveolocapillary CO₂ gradient.

Infrared monitors measure the absorption of infrared radiation of a particular wavelength, and monitor only one gas at a time. These devices are placed at the end of an endotracheal tube or a tight-fitting face mask. Two general types of analyzers are available — the breathe-through cell and the device that continuously aspirates end-tidal gas. If foreign material, including water, obstructs either the cell or the sampling line of the aspirating type, inaccurate measurements are obtained.

Mass spectrometers are expensive devices that can monitor up to eight gases simultaneously. One machine can also monitor several patients. By sampling, ionizing, and separating charged particles of a vapor mixture, the mass spectrometer can determine the concentration of a gas by its mass/charge ratio. Machines of recent design are versatile research tools, which can be used to monitor both inspired and expired CO₂, and thus facilitate determinations of minute volume, metabolic gas exchange, dead-space, functional residual capacity, pulmonary blood flow, and lung water.²

End-tidal CO₂ analyzers are useful as trend indicators in the management of patients with respiratory failure. ETCO₂ monitoring can provide a reasonably accurate index of alveolar ventilation during anesthesia, although cardiovascular depression or hypovolemia introduce discrepancies that limit its usefulness. The monitor is valuable for detection of air emboli during intracranial surgery. Weaning from mechanical ventilation is facilitated by ETCO₂ trending, particularly in patients with central respiratory depression who are subject to apneic episodes.

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Ear Oximetry

Spectrophotometric devices for measuring hemoglobin saturation have been used for many years, but only recently have noninvasive instruments been developed that are accurate, dependable, and easy to calibrate. The ear oximeter monitors the transmission of light through the pinna of the ear at two wavelengths, one where transmission of oxyhemoglobin and reduced hemoglobin are equal, and the other where the transmissions are widely divergent. By comparison of these values the percentage saturation of hemoglobin can be determined. The ear probe, which is heated to 37°C, is clipped to the pinna, and the local circulation is maximized by nicotine cream or by rubbing the skin. The new monitors internally calibrate to adjust for skin pigmentation, ear thickness, and movement of the earpiece.³ Both models have response times of less than 10 seconds, and thus are useful for detecting rapid changes in saturation.

In general, there is excellent correlation between SaO_2 derived from the ear oximeter and the oxygen saturation of brachial artery blood.⁴ Some discrepancies arise when the SaO_2 falls below 65 per cent, since small changes in PaO_2 correspond to large changes in SaO_2 . Ear oximetry may underestimate SaO_2 in patients with jaundice, while readings may be falsely high in patients with carboxyhemoglobin levels above 3 per cent. Also, since the ear oximeter measures only hemoglobin saturation it provides no information concerning PaO_2 tensions above 100 torr.

Ear oximetry can be employed to advantage in intensive care units to monitor changes in mechanical ventilator settings, to provide constant monitoring during suctioning and endotracheal intubation, and to furnish rapid feedback during trials of ventilator withdrawal. Other uses of ear oximetry include exercise studies in patients with chronic respiratory diseases,⁵ for sleep studies, and for bronchoscopic procedures. Its employment for continuous evaluation of oxygenation during anesthesia has also been suggested.⁶

Pulse Oximetry

The pulse oximeter determines the SaO_2 by a spectrophotometric method similar to that of the ear oximeter. The major difference is that the pulse oximeter, which fits around a finger, monitors SaO_2 only during the pulse wave, and in this way measures the saturation of the arterial blood. No heaters are required, and there is little interference from venous blood or skin. No external calibration is needed. There is good agreement between readings

of the pulse oximeter and *in vitro* oximetry readings between SaO_2 values of 55 and 100 per cent.⁷

A disadvantage of this method is that reduction of pulse volume diminishes the accuracy of the SaO_2 measurements. A mean blood pressure of less than 50 torr, vasoconstriction due to hypothermia or pressor drugs, or a pulse pressure of less than 15 torr may significantly curtail finger pulsations, although a special nasal probe can be used which is less affected by circulatory changes. Other variables that alter pulse oximeter readings are changes in finger flexion or extension, the presence of carboxyhemoglobin, and severe acidosis, where a shift of the oxyhemoglobin dissociation curve may introduce an error of as much as 10 per cent.

The pulse oximeter is easier to apply and more comfortable to the patient than the ear oximeter. These devices do not replace arterial blood gas analyzers, but they can be valuable for continuous monitoring of critically ill patients; they can warn of hypoxic events, and can provide useful indices for adjusting ventilator settings.

Transcutaneous Oxygen Monitoring

Measurement of the diffusion of oxygen through the normal skin was described 30 years ago. Heating the skin to render it hyperemic greatly increases diffusion, and provides a quantitative method for estimating both tissue oxygenation and perfusion. Transcutaneous oxygen tension, or P_{tcO_2} , is measured with a standard Clark electrode applied to the skin. The oxygen delivered to the epidermis depends on the PaO_2 , the oxyhemoglobin dissociation curve, and the cutaneous blood flow. To optimize blood flow, the electrode is heated to about 44°C, which induces hyperperfusion and maximizes local arterial dilation. The heat required per unit of time to maintain the electrode at a given temperature is continuously displayed. Because this is basically equal to the heat loss to the tissues one can use this information to estimate the changes in perfusion of the skin.⁸

Transcutaneous oxygen monitors have been used clinically for the past decade, particularly in newborn infants with respiratory problems.⁹ In the neonate the P_{tcO_2} is nearly equal to the PaO_2 , provided that peripheral circulation is normal. The skin of the adult is somewhat less permeable, and if optimally perfused yields a P_{tcO_2} reading about 80 per cent of the PaO_2 value. The two readings diverge increasingly as skin blood flow diminishes; during shock states the P_{tcO_2} more accurately measures perfusion than oxygenation of the blood.¹⁰ Oxygen delivery — the product of arterial oxygen content

and cardiac output — may be a more important parameter to monitor than PaO_2 , because it more accurately reflects the status of organ and tissue function.^{10, 11} Thus, $\text{P}_{\text{tc}}\text{O}_2$ monitoring does not obviate the need for arterial blood gases, but it can diminish the number of determinations required, and will furnish continuous information which can warn of impending problems, either respiratory or cardiovascular.

Since the skin at the monitoring site must be heated to about 44°C , there is some hazard of injury due to burns. The site should be changed every three to four hours. Because the number of cutaneous capillary loops per unit area varies from one site to another, the electrode must be calibrated every time the position is changed. This procedure becomes less laborious after it has become a routine; in large neonatal intensive care units $\text{P}_{\text{tc}}\text{O}_2$ monitoring is not a particularly burdensome addition to the workload. There is poor correlation between $\text{P}_{\text{tc}}\text{O}_2$ and PaO_2 during hyperoxia, which must be remembered when one considers the risk of retrolental fibroplasia in newborns.¹²

The transcutaneous oxygen monitor has many clinical applications. Its use in adults has tended to be more to measure tissue perfusion status.¹³ $\text{P}_{\text{tc}}\text{O}_2$ monitoring is a valuable adjunct in anesthesia management.¹⁴ PaO_2 estimates can be made in less than one minute by placing a drop of blood on the surface of a $\text{P}_{\text{tc}}\text{O}_2$ electrode.¹⁵ By applying the monitor to operative sites, plastic surgeons can determine the feasibility of transferring and separating skin flaps.¹⁶ Useful information on both oxygenation and perfusion can be obtained by monitoring during cardiopulmonary resuscitation.¹⁷ Thus, $\text{P}_{\text{tc}}\text{O}_2$ measurement has already achieved far-reaching utility.

Transcutaneous Carbon Dioxide Monitoring

Carbon dioxide is readily diffused through the skin, and can be measured with considerable accuracy by a Severinghaus electrode, which is an adaptation of the pH glass electrode. The $\text{P}_{\text{tc}}\text{CO}_2$ monitor can be obtained as an individual unit, or it may be combined in a single housing with a $\text{P}_{\text{tc}}\text{O}_2$ electrode. Although $\text{P}_{\text{tc}}\text{CO}_2$ is measurable at normal skin temperatures the monitoring site is usually heated to 44°C in order to decrease response time.

$\text{P}_{\text{tc}}\text{CO}_2$ values are significantly higher than the corresponding values of PaCO_2 . The gradient between skin and arterial blood is usually more than 20 torr,¹⁸ so that normal $\text{P}_{\text{tc}}\text{CO}_2$ values are commonly 60 torr or above. During shock states the CO_2 may be preferentially eliminated through the skin, and values of 100-125 torr may be reached.¹⁸ Experience

has shown that the $\text{P}_{\text{tc}}\text{CO}_2$ is a less reliable index of circulatory status than the $\text{P}_{\text{tc}}\text{O}_2$, and thus is less valuable as a monitor of cardiopulmonary decompensation.

The $\text{P}_{\text{tc}}\text{CO}_2$ analyzer has been frequently utilized to monitor CO_2 exchange in stable pediatric patients.⁹ Because anesthetic agents do not interfere with $\text{P}_{\text{tc}}\text{CO}_2$ sensor function, the device can be used during anesthesia to assess the adequacy of ventilation. Other applications include the monitoring of children with chronic upper airway obstruction,¹⁹ and monitoring fetal CO_2 outflow via a scalp electrode.²⁰

Conclusion

Five types of noninvasive monitors are described: the end-tidal CO_2 analyzer, the ear oximeter, the pulse oximeter, and transcutaneous monitors of O_2 and CO_2 . All of these provide useful information concerning respiratory and circulatory changes, but they may not correlate with PaO_2 or PaCO_2 values. Their chief advantage is the continuity of information they offer, which can help to identify untoward clinical events that are sudden and unexpected. When physiologic changes are occurring, these monitors can indicate when blood gases should be drawn. As technological refinements are added, these diagnostic instruments should achieve broader applicability in the future.

References are available from Dr. Mathewson, USKM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.

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Diabetes Mellitus and Viruses

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THE PATHOGENESIS OF diabetes mellitus, type 1 (acute onset or juvenile diabetes) will be considered in this review. Although non-insulin dependent diabetes (type 2 or adult onset) constitutes 80 per cent of all cases, viruses and autoimmunity are not considered to play a major role in its pathogenesis.¹ Type 1 is associated with three factors: viral infection, genetic vulnerability (connected with certain HLA antigen types), and autoimmunity (islet cell antibodies).

History

Since J. Stang of Norway reported diabetes following mumps infection in 1864, two significant factors have evolved. First, the seasonal incidence of juvenile diabetes has focused attention on those viruses that produce seasonal diseases. Second, the relationship between these seasonal incidence viruses and pancreatitis was reported in 1926 by S. Franklin Adams of the Mayo Clinic. Additionally, the picornaviruses,[†] prevalent in humans and seasonal in incidence, have been strongly suspected since 1950. Pancreatic disease has repeatedly been produced experimentally in mice with coxsackie B virus and a strain of encephalomyocarditis virus (EMC) was demonstrated in 1968 to cause diabetes in mice. Necrotic lesions in the pancreas of a newborn with coxsackie B type 4 virus infection were reported in 1956; subsequently, this virus was isolated from the pancreas of an infected newborn (1963) and an adult with acute pancreatitis (1964). Moreover, cattle have developed diabetes following hoof and mouth disease.

In the late 1960s in England, two studies confirmed the seasonal incidence factor in relation to viral antibodies of coxsackieviruses B types 1-6 as well as mumps and common respiratory viruses in diabetic patients. As is characteristic of enteroviral infection, a peak in autumn and significantly higher

antibody titers to coxsackieviruses B (especially type 4) occurred in type 1 diabetics more frequently than in normal subjects or those with diabetes of longer duration.^{2, 3}

However, a laboratory-proven epidemic of coxsackievirus B type 4 infection in Aleut residents of the Pribilof Islands in 1967 failed to confirm such factors. Paired pre-epidemic and convalescent sera of 117 persons showed 89 (77%) to have a four-fold or higher increase in antibody titer (two males, aged 46 and 58, had preexisting specific antibodies). In 1973, glucose tolerance tests of 136 persons under the age of 25 years produced normal values despite evidence of infection in 1967.⁴

Conflicting results were also obtained during the 1970s regarding the pathogenetic role of viruses (especially coxsackievirus B) in human and animal studies, but information regarding the causation of the disease has emerged as a result of these investigations.

Scope of Viral Agents

It has been noted that while juvenile diabetes is common, human pancreatitis from viruses listed above is uncommon, under natural conditions, with the exception of coxsackievirus B, thus prompting attention to the latter's role. Both animal models and humans with evidence of the disease have been studied. Despite the fact that coxsackievirus B infection in children is far more common than juvenile diabetes, continued reports of the disease-virus relationship from different areas focus on the virus as a possible etiologic agent.

Viral Pathogenesis and Pathology

Following the isolation of coxsackie B virus in 1948, a remarkable pancreatic acinar necrosis apparently sparing the islets of Langerhans and ducts was noted in suckling and weaned mice infected with it, the latter developing chronic pancreatic insufficiency, but none of the other coxsackie B virus lesions (*i.e.*, encephalitis, necrotizing steatitis). Demonstrated frequently with certain strains, this characteristic was lost by prolonged brain-to-brain passage of the virus.

From the EMC virus (first isolated from the myocardium of a pig dying of myocarditis), the M variant was produced in the late 1960s by repeated passage through mouse myocardium and was found

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† Polioviruses (3 types), coxsackieviruses A (24 types) and B (6 types), echoviruses (34 types), enteroviruses types 68-72, rhinoviruses (100 types), encephalomyocarditis (EMC) virus, and foot and mouth disease virus.

to produce necrosis of the insulin-producing beta cells. Mice thus infected developed clinical diabetes, and cortisone injection intensified islet damage. Acinar necrosis was absent but the beta cells, undergoing degranulation and contraction, exhibited alteration of cytoplasmic organelles followed by interstitial fibrosis with an autoimmune reaction suggested by the infiltration of lymphocytes and macrophages. While this diabetes mellitus-like reaction resulted only with the M variant in an inbred strain of male mice,^{5, 6} its production by certain newly isolated diabetogenic variants was prevented by interferon and a variety of interferon inducers, suggesting that interferon's protective effect is probably genetically controlled.⁷

Viral Etiology

Thus the role of coxsackieviruses B in the etiology of juvenile diabetes is supported by the results of animal model studies, predilection of the viruses for pancreatic tissue, and isolation of the viruses from the infections temporally associated with onset of the disease.

Serologic evidence of this third factor developed in 1977 when high-titer neutralizing antibodies to coxsackievirus B type 2 in acute onset diabetes in an 18-month-old boy were documented.⁸ Coxsackievirus B type 4, indicated by a rise in specific antibody, was recovered from the pancreas of a 10-year-old dying from a flu-like illness in 1979; lymphocytic infiltration of the islets and beta cell necrosis were found at postmortem examination.⁹ An inbred strain of mice injected with the isolated virus developed hyperglycemia, inflammatory cells in the islets, and beta cell necrosis.

More recently, coxsackievirus B type 5 was isolated from the stools of a 16-month-old child with an episode of fever and acute thrombocytopenic purpura. Serological tests confirmed the involvement of the isolated virus, and laboratory evidence for diabetes mellitus was obtained on days 13 to 23. A two-month remission followed, after which definitive juvenile diabetes developed. The child had high-risk genetic markers for diabetes, and developed islet-cell antibodies one week prior to onset. The isolated virus showed pancreatropism for mice. Thus viral, genetic, and autoimmune factors were believed to have been involved in this case.¹⁰ A seroepidemiological study of 28 children with type 1 diabetes which developed in Britain in 1982 indicated that coxsackieviruses B, particularly type 4, were associated with this disease. Recognizing the involvement of familial tendency and HLA antigens DR3 and DR4, the investigators suggested that perhaps infection with coxsackieviruses B triggers

the onset of the clinical manifestations.¹¹

Despite such evidence, serologic surveys indicate about one-half of the human population has been infected with coxsackievirus B, but juvenile diabetes develops in less than 0.1 per cent. Conversely, many cases of juvenile diabetes show no evidence of recent coxsackie B infection.

Since this review focuses on the role of viral infection, the other two factors cited — genetic vulnerability and autoimmunity — are not within its range. Autoimmune responses, however, cannot be totally excluded since viruses may trigger them, and beta cell susceptibility to diabetogenic viral effect may be enhanced by autoimmune response. As noted, only coxsackievirus B infections appear to be implicated. Their frequency and the rarity of juvenile diabetes suggest the requirement of an intrinsic property to determine diabetogenicity. Such has been noted in the M variant of EMC (1968) and repeated passage of other viruses in beta cell cultures produces in them a diabetogenicity for mice. Although random mutation is not ruled out, a selection for intrinsically diabetogenic strains is suggested.

More recently, an infant with myocarditis and pancreatitis provided a single isolate of coxsackie B type 4 with various strains showing biologic differences. Three of these and the original isolate were serologically similar, had identical plaque morphology and grew similarly in mouse pancreas, but differed in the number of islet cells infected and levels of virus antigen concentrated in them. Degranulation of beta cells and antigen accumulation were positively correlated in histopathologic preparations. Only the original isolate brought a significant increase in plasma amylase levels in infected mice.^{12, 13}

Thus heterogeneity within a single human virus isolate may produce strains with different biologic and pathogenic properties (since the mice used were genetically similar), and a strain endowed with predominantly diabetogenic property may produce the disease in genetically vulnerable individuals. The rarity of the disease therefore reflects the rarity of diabetogenic strains among the various virus types.

Multiple viral infections with cumulative beta cell damage have been proposed as a possible mechanism, the disease occurring when a critical level of damage has been done, but this concept seems less plausible than the one cited.¹⁴

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CNS Irradiation in Small Cell Lung Cancer

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SMALL CELL lung cancer (SCLC) accounts for about 20 per cent of all bronchogenic cancers.¹ It differs from other lung cancers in its propensity for rapid metastasis. Common sites include liver, bone, bone marrow, and brain.² Combination chemotherapy has prolonged the median survival to about 14 months,³ and some have reported a 25 per cent two-year survival.⁴ Patients who present with disease limited to the chest subsequently develop brain metastasis in 20-25 per cent of cases.⁵ Autopsy studies, however, reveal a much higher percentage of brain involvement,⁶ usually in the form of multiple intracerebral metastases, while about 10-15 per cent of patients develop leptomeningeal and epidural lesions.^{2, 7, 8} The median time to develop brain metastasis is about 16-18 weeks,⁹ and the possibility increases with longer survival. By 18 months, about 75-80 per cent of patients will develop brain relapse.^{10, 11} Once brain metastases develop, the survival rate is poor; most patients die within three months.^{12, 13} A majority of these patients will die of uncontrolled central nervous system (CNS) disease.¹⁴ Brain metastases in patients on chemotherapy is attributed to inadequate drug concentration with the CNS.

Prophylactic Irradiation

The logic for use of prophylactic irradiation for subclinical SCLC stems from the extensive use in acute lymphocytic leukemia, which also has a high risk of CNS involvement.¹⁴ In SCLC, a dose of 2,500-3,000 rads administered to the whole brain after induction chemotherapy, during a two-week period, decreased brain relapses from 25-30 per cent to less than 5 per cent in many studies. However, skepticism concerning the use of prophylactic irradiation has resulted from the observation that overall survival has not been extended.¹⁵⁻¹⁸

In evaluating benefits from prophylactic whole brain irradiation, two facts must be considered. First, in order to produce improvement in survival, systemic disease must be controlled. Unfortunately, the time when brain metastasis appears maximal in SCLC is also the time when systemic relapse occurs. Thus, improvement in survival is not seen.

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Second, only those patients who have prolonged control of systemic disease and have brain relapse as the only site of failure are likely to benefit from brain irradiation. However, both clinical and autopsy studies have shown that such failures occur in fewer than 6 per cent of cases; consequently, no significant survival benefit is seen.¹⁹⁻²¹

This, however, does not negate the fact that prophylactic irradiation does prevent CNS disease in a significant proportion of patients and, by this preventive effect, demonstrates a distinct superiority over therapy after brain disease is established.²² Furthermore, studies indicate an improvement in quality of life²³ without significant CNS changes.²⁴ Prophylactic whole brain irradiation is therefore recommended for all patients with limited disease who have responded to chemotherapy. An optimum time for CNS prophylaxis has not been determined, but it is probably best given up front after two to three cycles of chemotherapy.

Therapeutic Irradiation

About 10 per cent of patients with SCLC present with brain metastasis, and about 5 per cent of patients who receive prophylactic brain irradiation develop CNS relapse.²⁵ A dose of 3,000 rads in two weeks to the whole brain offers adequate palliation. Higher doses do not necessarily increase the number or duration of responses.²⁶ The only patients who may benefit from a higher dose of 4,000 rads in a three to four week period are those who have CNS relapse with systemic disease under control. Improvement in neurological function is prompt and can be seen in approximately 50 per cent of patients with some specific neurologic signs of improvement in as many as 90 per cent.

Leptomeningeal Involvement

Clinically, about 10 per cent of patients can develop involvement of the meninges, either alone or in combination with intracranial metastasis. Treatment usually consists of whole brain irradiation and intrathecal methotrexate. The objective response as measured by the clearing of tumor cells in the cerebrospinal fluid has been reported to be 40-80 per cent.²⁷ Craniospinal irradiation is another alternative method for treatment of these patients. However, most have been heavily pre-treated with chem-

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Postmenopausal Osteoporosis

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POSTMENOPAUSAL osteoporosis is becoming increasingly important because of the growing numbers of women living well beyond menopause. Osteoporosis is associated with significant morbidity and mortality. In postmenopausal women, it can be demonstrated in one out of three, and at least 20 per cent will suffer a hip or vertebral crush fracture by the age of 90 years.¹ Osteoporosis can be demonstrated in 80 per cent of patients with hip fractures, and 34 per cent of patients with hip fracture die within six months.² It has been estimated that each year, in the United States, about 120,000 elderly women incur hip fractures, and that about 18,000 will die as a result.³ Osteoporosis occurs whenever rates of bone formation are lower than rates of resorption. The rate of formation is approximately equal to the rate of resorption until age 40 years, when resorption is greater than formation, especially in women. Osteoporosis is characterized by bone composed of smaller than normal trabeculae and cortex, which causes structural weakness and increases the risk of fracture. Women lose bone faster than men, and the rate of loss increases markedly at menopause. The increase in fasting-plasma and urine calcium levels that accompany natural menopause and oophorectomy, seems to indicate that loss of ovarian function and osteoporosis may be causally related.⁴

Postmenopausal osteoporosis seems to have a multifactorial genesis. The decrease of the inhibition of osteoclastic bone resorption, which is estrogen dependent, appears to be one important factor. However, many other factors, such as poor calcium intake, decreased intestinal calcium absorption, hypercalciuria, decreased production of vitamin D, decreased physical activity, and increased parathyroid hormone effect, may all be additional important factors.¹ Some of these factors are reviewed briefly in the ensuing paragraphs.

Estrogen Deficiency. Estrogen deficiency, as indicated above, appears to play an important role in the development of osteoporosis. Bone mass and estrogen secretion decline in parallel after the menopause. Studies by Heaney *et al.*⁵ showed that bone resorption occurs at a rate exceeding that of bone formation after menopause, and that estrogen therapy reduced bone calcium resorption and improved calcium balance.

Calcium Intake. Reduced calcium intake is another important factor in the development of postmenopausal osteoporosis. Several studies have shown that treatment of osteoporosis with oral calcium supplements decreases age-related bone loss. Riggs and associates⁵ showed by bone biopsy that a high calcium intake reduced bone resorbing activity; they attributed the favorable response to suppression of endogenous parathyroid hormone secretion. Heaney's work⁵ showed that postmenopausal women have a calcium requirement of 0.990-1.504 g/d, and that the actual average intake is 0.480 g/d.

Calcium Absorption. A common finding in postmenopausal osteoporosis is decreased calcium absorption, which also contributes to negative calcium balance. Approximately 30 per cent of osteoporotic women have decreased intestinal calcium absorption. Studies by Gallagher *et al.*⁷ showed calcium absorption to be related to estrogen levels and the vitamin D endocrine system. Osteoporotic women were placed in three treatment groups — placebo; 1.25-2.5 mg/d of conjugated equine estrogens; and 0.5 mg synthetic 1,25-dihydroxyvitamin D₃ — and were evaluated before and after six months of therapy. Fractional calcium absorption was unchanged after treatment with placebo, but was increased in the groups receiving estrogen and 1,25-dihydroxyvitamin D₃. Serum 1,25-dihydroxyvitamin D₃ and serum parathyroid hormone were increased after treatment with estrogen, but not after placebo treatment. This study tends to indicate that estrogen treatment increases calcium absorption in postmenopausal osteoporotic women by increasing serum 1,25-dihydroxyvitamin D₃.⁶ Prospective studies evaluating the effectiveness of the administration of dihydroxyvitamin D₃ are underway, but results are not yet available.

Parathyroid Hormone. Parathyroid hormone (PTH) is thought to play a role in postmenopausal osteoporosis. PTH governs osteoclastic activity, causing progressive bone loss and osteoporosis. Wiske and co-workers⁸ showed increasing PTH levels with advancing age after 40 years — more so in women than men — and this increase was associated with a significant decrease in serum ionized calcium and inorganic phosphate.

Age. Age is another factor which contributes to the pathogenesis of osteoporosis. Bone mass de-

creases as age increases after the fourth decade. The rates of bone loss are estimated to be 4 per cent per decade in men and 8 per cent per decade in women.¹ Several age-related events cause a decrease in serum ionized calcium and a secondary increase in PTH secretion, which may contribute to osteoporosis. Advancing age may be associated with an impairment of hepatic hydroxylation of vitamin D, inability to increase intestinal calcium absorption, and reduce renal production of 1,25-dihydroxy-vitamin D.

Early identification of osteoporosis is difficult because it develops gradually and symptoms are not easily identified until the disease has progressed. Increased radiolucency, as seen on conventional x-ray films of the vertebrae, ribs and femoral neck, is not detected until about 35 per cent of bone mass has been lost. Measurement of cortical and trabecular density using photon absorption is an accurate method for detecting osteoporosis if it is used sequentially every two years; however, it is not a practical routine clinical tool.¹ Other parameters which are useful in the early identification of osteoporosis include: elevated fasting urinary calcium/creatinine ratio; elevated fasting urinary hydroxyproline/creatinine ratio (> 0.012); plasma progesterone levels less than 0.5 ng/ml; elevated PTH plasma levels; low plasma calcitonin levels; and marked loss of height.³

Therapy of Osteoporosis

Various therapeutic regimens have been studied for the treatment of osteoporosis, but the most effective seems to be the administration of estrogens. In a ten-year study by Nachtigall and co-workers,⁹ 84 matched pairs of hospitalized postmenopausal women who were at high risk for osteoporosis because of limited physical activity, were divided into two groups. One group received placebo and the other group received conjugated equine estrogens, 2.5 mg/d, and medroxyprogesterone acetate tablets, 10 mg/d for seven days of each month. All patients received 2 g of dietary calcium per day. Each group was subdivided into those who had their last menstrual period within three years and those who were beyond three years of menopause. *Figure 1* shows the changes in bone mass as determined by photon absorptiometry and calculated and graphed as the linear absorption co-efficient before and after ten years. Patients in the placebo groups lost bone mass gradually during the ten year period, while patients in the hormone-treated groups either gained bone mass or experienced minimal bone loss. This study clearly demonstrates that estrogen therapy is effective

DENSITOMETRIC LINEAR ABSORPTION COEFFICIENTS

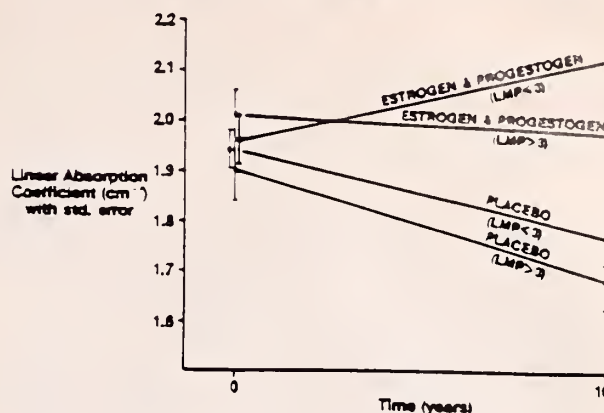


Figure 1. Densitometric linear absorption coefficients in treated patients with LMP \leq three years, treated patients with LMP $>$ three years, control patients with LMP \leq three years and control patients with LMP $>$ three years. (Adapted from Nachtigall *et al.*, with permission of the authors and reprinted with permission of The American College of Obstetricians and Gynecologists, OBSTETRICS AND GYNECOLOGY, Volume 53, Number 3, March 1979, page 278.)

to prevent osteoporosis. Since every patient in this study received adequate dietary calcium, it can be concluded that calcium without hormonal therapy will not protect against osteoporosis.

Christiansen *et al.*¹⁰ have shown that even temporary hormone replacement with estrogen will have a persistent, beneficial effect on bone mass and that estrogen therapy is beneficial in preventing the progression of osteoporosis. They studied the effects of initiation and withdrawal of estrogen therapy on forearm bone mineral content (BMC) in a randomized trial of 94 healthy female volunteers six months to three years after menopause. Group A, which received estrogen therapy for three years, had an increase in BMC (measured by photon absorptiometry) of 3.7 per cent, while in the placebo group (Group B), the BMC decreased by 5.7 per cent. After 24 months, the groups were subdivided: Group Aa continued hormone therapy, Group Ab discontinued hormone therapy and received placebo, Group Bb continued placebo, and Group Ba started on hormone therapy. The annual rate of bone loss after discontinuation of hormone therapy was identical to the bone loss observed in the placebo group. The results are summarized in *Figure 2*. They concluded that, in postmenopausal women, even temporary hormone replacement had a lasting beneficial effect on bone mass.

Geola *et al.*¹¹ studied the effects of various dos-

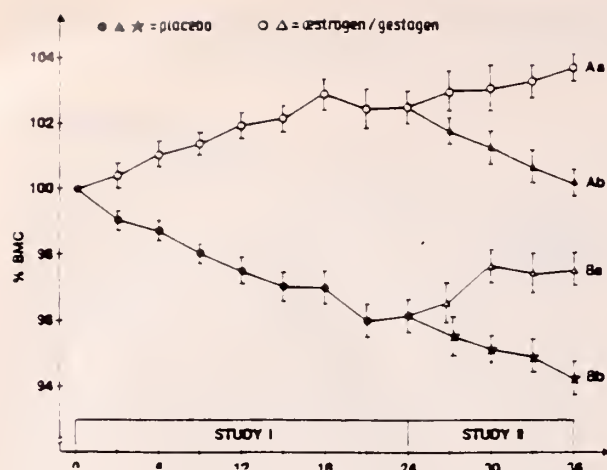


Figure 2. Bone mineral content as a function of time and treatment in 94 (Study I) and 77 (Study II) women soon after menopause. (From Christiansen *et al.*,¹⁰ with permission of the authors and reprinted with permission of the publisher.)

ages of conjugated equine estrogens to correct postmenopausal symptomatology, such as hot flashes, atrophic vaginitis, and osteoporosis. They concluded that a dosage of 1.25 mg was necessary for suppression of hot flashes and for the correction of atrophic vaginitis, and 0.3 mg for the prevention of osteoporosis as measured by correction of the calcium/creatinine urine ratio. However, prospective, randomized studies are necessary to substantiate the latter statement.

Additional therapeutic factors are calcium intake, balanced diet, and exercise. Adequate calcium intake is also necessary to prevent osteoporosis. The body requires 1,000 mg of elemental calcium per day and the average, usual intake is only 600 mg, so supplementation with calcium salts is usually necessary. In addition, adequate supplies of vitamin D are necessary to insure intestinal absorption of calcium. In most women, these supplies are available. However, due to diminished renal function with aging, a reduced conversion of hydroxyvitamin D to dihydroxyvitamin D could occur. If such a deficiency is suspected, or in patients with poor dietary

habits or aversion to sunlight, vitamin D should be given.¹

Prevention of Endometrial Cancer

Numerous studies have revealed an increased incidence of endometrial cancer in women receiving estrogens alone, as compared to matched controls not receiving estrogens. The relative risk has ranged from three to nine. This risk is dose related and the lower the dose, the lower the risk. Studies have shown that the development of endometrial hyperplasia and carcinoma in women receiving estrogen therapy can be prevented by the administration of cyclic progestational therapy.^{12, 13} In a prospective study¹² of 745 women, estrogens were administered at various dosages, alone or in combination with progestational agents at various dosages and for a different number of days every month. Endometrial curettage was performed at six months and yearly thereafter for a total of 21,736 months-study. No patients developed endometrial hyperplasia or carcinoma when a progestogen was given for ten or more days on a monthly basis. This study also revealed a lower incidence of endometrial hyperplasia and carcinoma in women receiving low-dose estrogen as compared to those receiving high-dose estrogen.

In conclusion, osteoporosis will develop in an important number of postmenopausal women and is associated with significant morbidity and mortality. Estrogen therapy has been proven to be effective for the prevention and retardation of osteoporosis. Estrogen therapy alone is associated with an increased incidence of endometrial malignancy, and this incidence is dose related. When estrogens are given with a progestogen for at least ten days every month, endometrial malignancy is prevented. It seems that the administration of conjugated equine estrogens 0.3 mg/day (21 days), and a progestogen (medroxyprogesterone acetate 10 mg) for days 11 to 21 in selected postmenopausal patients, should be effective in the prevention or retardation of osteoporosis without an increased risk of endometrial malignancy.

References are available from Dr. Magrina, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.



Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

AIDS — Practical Guidelines

NABIH I. ABDOU, M.D., Ph.D.,* *Kansas City, Kansas*

THE CLINICAL syndrome of acquired immunodeficiency (AIDS) has received extensive attention since the first cases were reported in 1981. The marked social, epidemiological, and clinical implications of AIDS make it imperative that the practicing physician be aware of its current concepts. Excellent reviews and updated comments have been published recently.¹⁻³

Criteria for the diagnosis of AIDS are outlined in *Table I*. Although those criteria are sufficient for epidemiological studies, they do not include the patient with early features of AIDS, namely lymphadenopathy and constitutional symptoms present prior to development of any opportunistic infections or Kaposi's sarcoma.

Epidemiology

More than 2,000 cases have been reported nationwide since 1981. The numbers have doubled every six months. Cases have been reported predominantly in large cities on the east and west coasts. Very few cases have been reported from the Midwest. AIDS patients were shown to be homosexuals in 71 per cent of the cases; intravenous drug abusers, 17 per cent; Haitian immigrants, 5 per cent; hemophiliacs, 1 per cent; miscellaneous, 6 per cent (female sexual partners of AIDS patients, infants in high risk populations). About one-third of the reported cases have been fatal. Most common life-threatening associations are *Pneumocystis carinii* and Kaposi's sarcoma.

From the Department of Medicine, Division of Allergy, Clinical Immunology and Rheumatology, The University of Kansas School of Medicine-Kansas City.

* Professor of Medicine.

Address reprint requests to Dr. Abdou, UKSM-KC, Room 4035B, 39th & Rainbow Blvd., Kansas City KS 66103.

Modes of Presentation

Patients with possible AIDS may present with the various features outlined in *Table II*. These include lymphadenopathy, prodromal symptoms, opportunistic infections or Kaposi's sarcoma. In many patients presenting with lymphadenopathy and prodromal symptoms, workup for lymphoma, including lymph node biopsy, is indicated.

Opportunistic infections may be viral, fungal, protozoal, mycobacterial, or others (*Table III*). Viral infections include cytomegalovirus and herpes simplex; the former frequently can be cultured from urine and sputum. Herpetic infections are often perianal or periorbital. Infection with *Candida albicans* is frequently seen in AIDS and may be either limited to oral or esophageal mucosa or may be disseminated. *Cryptococcus neoformans* meningitis or the disseminated form have been reported. Several protozoal infections have been detected in AIDS. *Pneumocystis carinii* pneumonia carries a serious prognosis. *Toxoplasma gondii* encephalitis and *Cryptosporidium enteritis* are occasionally seen. Both *Mycobacterium tuberculosis* and *Avium intracellulare* have been reported, and cultures for these organisms should be requested. Other unusual infections include nocardia and Legionella.

TABLE I
ACQUIRED IMMUNE DEFICIENCY SYNDROME
(AIDS): DEFINITION

- Life-threatening opportunistic infection
OR
 - Kaposi's sarcoma
- In a person younger than 60 years old
No underlying immunosuppressive disease
No prior immunosuppressive therapy

TABLE II
AIDS: MODES OF PRESENTATION

- *Lymphadenopathy*: Generalized, prolonged, unexplained
Inverted T helper:T suppressor ratio
Absence of opportunistic infections
- *Prodromal phase*: Fever of unknown origin, weight loss, lymphadenopathy
- Opportunistic infections*
Characteristic laboratory features:
Lymphopenia
Cutaneous anergy
Inverted T helper:T suppressor ratio
Poor in vitro response to mitogens
- Presentation with either of the above plus *Kaposi's sarcoma*

Characteristic Laboratory Features

There are no diagnostic laboratory tests for AIDS. However, a patient with a suggestive clinical picture and who has any of the following laboratory abnormalities should be suspected. These include lymphopenia, cutaneous anergy and inverted T helper to T suppressor ratio. Cutaneous anergy should be elicited by applying delayed skin tests with at least five antigens: tuberculin, mumps, trichophyton, candida, and histoplasmin. The T helper (T4) to T suppressor (T8) ratio normally is > 1.8 . In AIDS, it is usually < 1.2 . Apparently ratios < 0.6 are seen in patients with opportunistic infections or Kaposi's sarcoma. The inverted T helper to T suppressor ratio is caused by a decrease in T helper cells.

Possible Etiologic Mechanisms

The etiology of AIDS is still unknown. Epidemiological data indicate that a transmissible infectious agent could play a role, but the nature of the infectious agent is still unknown. Various viruses are suspected, but their role in AIDS is still unproven. These include human retrovirus (human T cell leukemia virus), mutated cytomegalovirus, and Epstein Barr virus. Failure of immune mechanisms leading to compromise of host defenses is an essential predisposing factor. Recreational drugs and antigenic overload (spermatozoa, anti-hemophilia factor) have been shown to suppress the immune system. The suspected viral infectious agent seems to be lymphotropic with an affinity to T helper cells. Depletion of T helper cells results in the failure of production of both a lymphokine called interleukin-2 and interferon. Both interleukin-2 and interferon are essential in killing of virus-infected cells. These immunological findings explain the inverted T4:T8 ratio in patients with AIDS, inability of their cells to

TABLE III
AIDS: INFECTIOUS
COMPLICATIONS/ASSOCIATIONS

- Viral: Cytomegalovirus, Herpes simplex
- Fungal: *Candida albicans* (oral, esophagitis, disseminated)
Cryptococcus neoformans (meningitis, disseminated)
- Protozoal: *Pneumocystis carinii* (pneumonia, retinal)
Toxoplasma gondii (encephalitis)
Cryptosporidium (enteritis)
- Mycobacterial: *Avium intracellulare*
Tuberculosis
- Others: Nocardia, Legionella

produce interferon. This information also suggests guidelines for possible modulation of the immune system of AIDS patients. Although not widely confirmed, some AIDS patients show an increased incidence in the genetic marker HLA-DR5. Sociological factors also play a role in predisposing patients to AIDS, including promiscuity, use of recreational drugs, and sexual practices leading to rectal abrasions. Therefore, it is currently assumed that the etiology of AIDS is multi-factorial and includes a possible infectious agent, distortion of the immune system, a predisposing genetic background, and sociological factors. Investigators are concentrating on determining the sequence of events in the induction of AIDS and the nature of the infectious agent.

Prevention and Therapy

At present, isolation of patients with AIDS is not necessary. However, careful handling of secretions, blood or blood products from patients with AIDS should be done as in handling the same materials from hepatitis patients.

The physician should inform individuals who have or are suspected to have AIDS of the preventive public health recommendations outlined in *Table IV*.

Blood banks and hospitals across the country have established strict guidelines that require interviewing blood donors for evidence of early features of AIDS, proper handling of blood collected from known or suspected AIDS patients, and screening of blood donors for antibody to hepatitis B core antigen. The latter is considered by some to be a surrogate marker for AIDS. The following groups should refrain from donating blood: any person with clinical features of AIDS, homosexuals, bisexuals with multiple partners, recent Haitian immigrants, past or present intravenous drug abusers, and sexual partners of individuals at increased risk.

(Continued on page 96)



The Image

The contribution of narcissism to the medical personality is a risky one to pursue in a medical periodical but our conviction of its fundamental role exceeds our judgment. Before the hackles rise to a point of dislocation, we hasten the qualification that we are referring (mostly, anyway) to the so-called secondary form of narcissism which comes from a merging of the ego with the superego. Although the latter in this case is that idealized medical practice we all aim for, it can be argued that the condition is not confined to the medical profession but can be attributed to any pursuit warranting high dedication and commanding a certain social prestige — only that the nature of medical service intensifies the performance beyond others. Thus emboldened, we dare to extend the qualification to say that no discipline, whatever the justification, is more imbued with admiration of its self-image than medicine.

The condition is manifested (for openers) in the age-old but now threatened conviction that the physician knows best, an essential element of medical service — if it doesn't get out of hand. Medical authority sees this conviction as benefiting the patient as much as the physician's ego, and it can be altogether commendable if it isn't contaminated with the primary narcissism of self-love. The recurrent charge of medical arrogance is an inevitable by-product when the patient can't distinguish between the two in the physician's behavior, especially in these days of medical populism.

The same concept emerges on the economic level in the fee-for-service tradition since this embodies not only the physician's control over what service will be rendered but what its value should be in market-place terms. Nothing in medical practice today is under more relentless attack, not just the determination of the monetary value but whether the physician's professional decision of appropriate service is valid economically — and by extension, professionally.

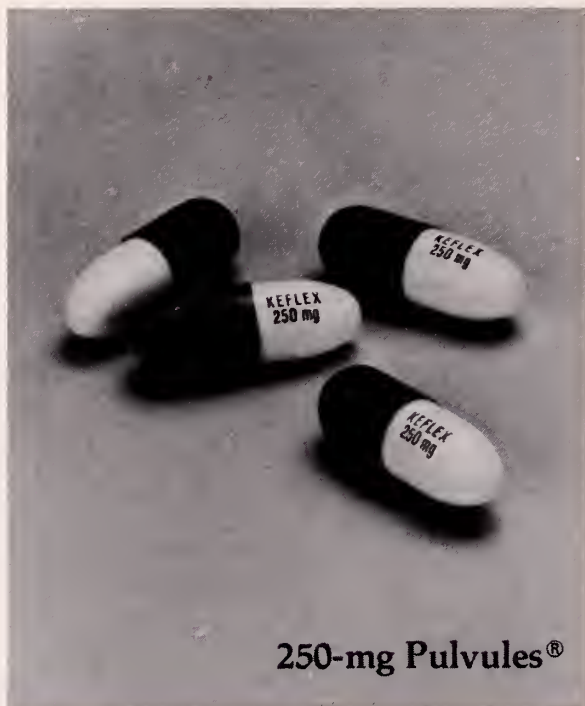
This brings us to two features of the current medical revolution in which the pragmatic aspects

have obscured the impact on the physician's self-image (again read narcissism). First, the numerous plans currently being promoted to produce an affordable medical structure have certain features in common despite surface differences. Although medical advisers may be a part of the structure or integral in its function, the forces determining and regulating the function are increasingly non-physician. This is not to impugn the intent or integrity of all lay persons involved but they are, of necessity, approaching the problem as one of economics into which the medical elements will be introduced in, to them, appropriate form. At this point, protocols for the most part are careful to advise physicians that they may continue their usual charges — except as remuneration is regulated by provisions of the plan. The ultimate effect, of course, is expected to be the accommodation of physician-determined fees to the prevailing economic requirements, whether in or out of the plan — necessitating, it would seem, a consequent adjustment of the physician's self-image to the economic role.

But the traditional self-image is also under undeclared attack from the corollary shifting of the physician's relationship with other medical personnel and institutions. The increased use of ancillary personnel, initially (and still) fostered by the well-known factors of technology and physician availability, has moved from a comfortably physician-dominated system to one in which physicians must make increasing accommodation to this segment of medical care. There can be little doubt that physicians, whatever the professional justification for retaining firm control over the group, are additionally motivated by a reluctance to accept a diminished status. It means that a successful reformation of medical service will depend not only on the physician's ability to establish firm professional credentials but the production of a climate of mutual self-respect — and the patient's understanding of the relationship.

Well, stones landing in the pond would obviously agitate the surface and distort the image Narcissus has so enjoyed contemplating. Since his preoccupation with that image was in itself, however, a punishment, he might well wonder whether the stones were being cast at the pond or at him. — D.E.G.

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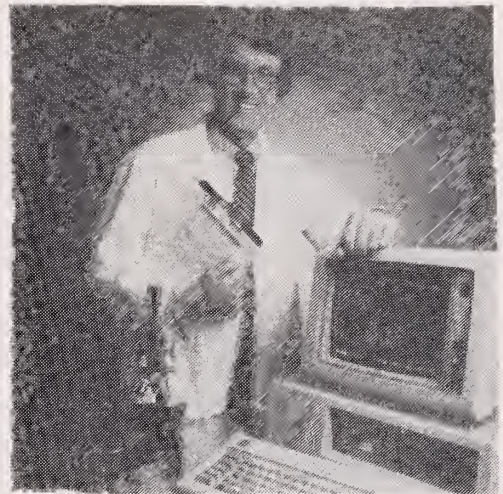
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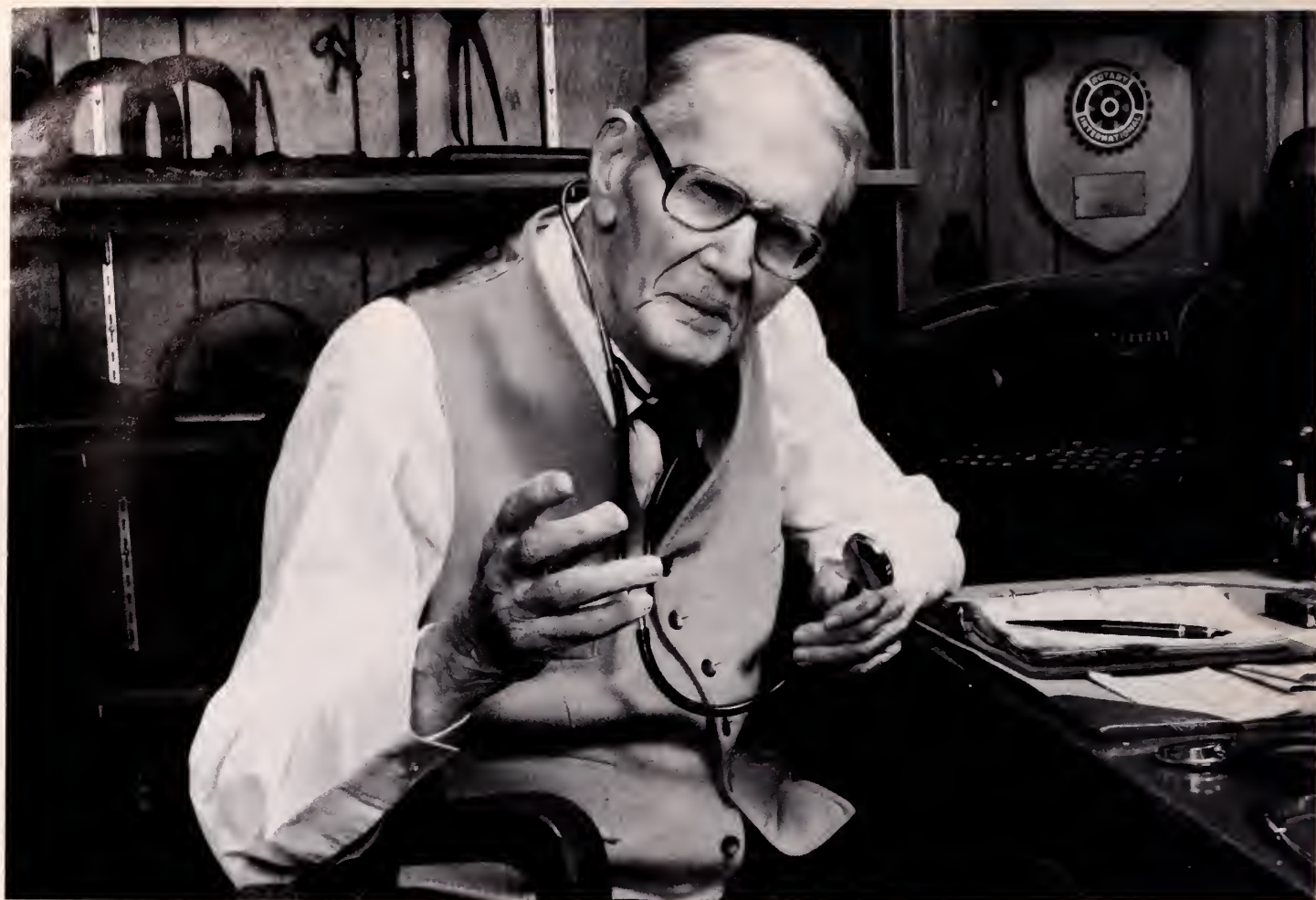
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Diagnostic Cholescintigraphy

(Continued from page 73)

The results of the scans on the 62 patients with possible acute cholecystitis are presented in *Table I*. Fifteen of the 17 true positive studies were surgically proven. Only two patients were managed medically and failed to have a tissue diagnosis. Nine of the 42 true negative patients had exploratory laparotomies and demonstrated the absence of acute cholecystitis. Clinical followup revealed no later gallbladder complaints in those 42 patients. There were two false positive examinations and one false negative examination.

Discussion

The sensitivity and specificity of cholescintigraphy performed at the Medical Center were 94 and 95 per cent respectively. These results are somewhat less than usually reported in the literature⁷⁻¹⁴ where pooled results from eight studies yielded a true positive rate of 98 per cent (428/438) and a true negative rate of 93 per cent (470/504). The true positive and true negative figures are quite impressive in view of the noninvasiveness of the test, the ease of performance, and general availability. Sonography can also make a claim to high sensitivity and specificity, but not as high as is usually quoted for cholescintigraphy.

On occasion, cholescintigraphy will identify

chronic cholecystitis as acute, but this discrepancy is not considered serious. The testing must be carried out within the prescribed time frame (2-24 hours after eating) to assure maximum accuracy.

We have obtained correct estimates of hepatocellular function and have excluded the diagnosis of common duct obstruction and the presence of acute cholecystitis even when the serum bilirubin was greater than 12 mg/100 ml. In one patient with gall stones, a bilirubin of 15 mg/100 ml and pancreatitis, visualization of the gallbladder was of great value in excluding acute cholecystitis and preventing exploratory laparotomy.

Cholescintigraphy has proven to be safe, sensitive, and specific. It usually takes only one hour to exclude the possibility of acute cholecystitis. The test is considered to be a routine examination and should be available in any nuclear medicine unit.

References are available from Dr. Martin, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.

CNS Irradiation

(Continued from page 79)

otherapy and may not have enough bone marrow reserve to withstand craniospinal irradiation. These patients in general do poorly and have a median survival rate of about two months.

In summary, considerable progress has been made in the management of SCLC. With the current techniques of chemotherapy, patients with disease limited to the chest who respond to chemotherapy should receive prophylactic whole brain irradiation early in the management. Patients with advanced disease who either present with or later develop brain metastases can be effectively palliated with a dose of 3,000 rads to the whole brain.

References are available from Dr. Giri, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.

TABLE I

	Clinical Truth	
	Acute Cholecystitis	No Acute Cholecystitis
Abnormal scan	17	2
Normal scan	1	42

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VOX DOX

Vox Dox Editor:

A letter to Vox Dox in the January *Journal* suggests that KMS wastes large amounts of money through its support of KaMPAC, helping destroy "the political system which has worked so well to make this country great" by weakening the political parties and corrupting politicians "(in a way)." In fact PACs sprang up because of party weakness and laws restricting party power.

The \$4,000 granted by KMS this year to KaMPAC is as always to be used for administrative purposes, such as postage, paper (letter type), and political education. In the latter category, KaMPAC actually supports the *Journal* because it pays for all PAC advertising published in it — \$1,430 during

1983. This advertising suggests that KMS members become active in three ways: first, by serving as committee members, delegates, and officers in KMS; second, by supporting their chosen candidates and parties with work, money, and votes; and finally by joining KaMPAC in its effort to identify and elect friends of medicine on behalf of our patients and ourselves.

The remainder of the money used by KaMPAC is raised through purely voluntary contributions and does not adversely affect the *Journal*, 1,000 calorie version or otherwise.

Thus, KaMPAC helps add effectiveness as KMS seeks to shape legislation and health policy for the benefit of all Kansans.

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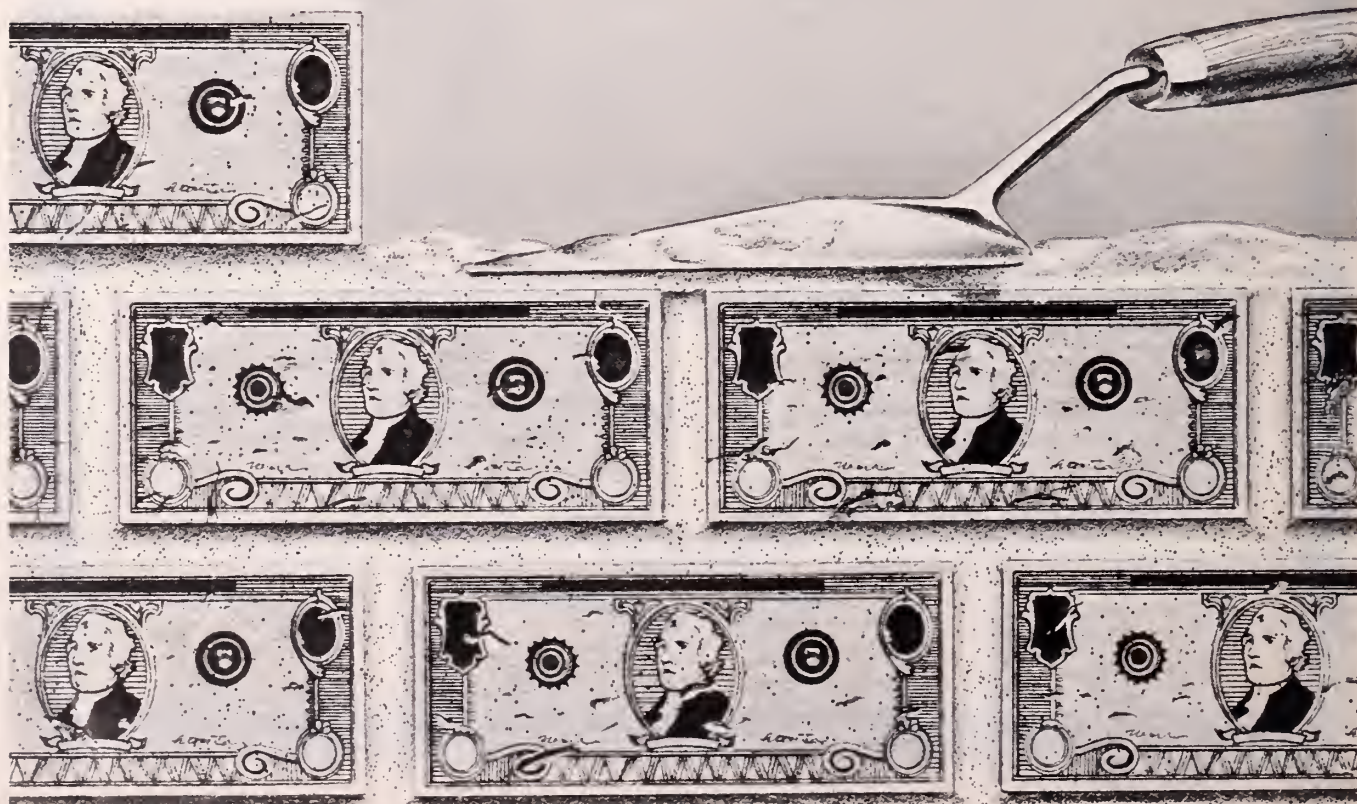


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AIDS

(Continued from page 84)

There is no specific therapy for AIDS, but aggressive early treatment of infections is absolutely indicated. Although serial followup of the T4/T8 lymphocyte ratio and delayed skin testing could theoretically be of value, there are no reports to indicate the reversal of the various immune abnormalities to normal upon clinical improvement.

At the present time there are several experimental therapeutic trials underway. These are directed toward achieving immune modulation by the administration of interferon, interleukin-2, or thymic hormones. Results of these trials are not yet available. Experience at UKSM seems to indicate that patients with prodromes of AIDS may be helped by prophylactic antibiotics such as trimethoprim-sulfamethoxazole and ketoconazole drugs.

The practicing physician should be aware of the features of AIDS, particularly those appearing early

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TABLE IV
AIDS: PUBLIC HEALTH RECOMMENDATIONS

- Sexual contact should be avoided with persons known or suspected to have AIDS.
- Members of high-risk groups should
 - I. Be aware that multiple sexual partners increase the probability of developing AIDS.
 - II. Refrain from donating plasma and/or blood.
- Physicians should adhere strictly to medical indications for transfusions.
- Autologous blood transfusions are encouraged.

in the course of the disease. Physicians should consult clinical immunologists for proper tests to support the diagnosis. They should be aware of and follow public health guidelines, and be aggressive in treating the several infections that can be devastating to the patient.

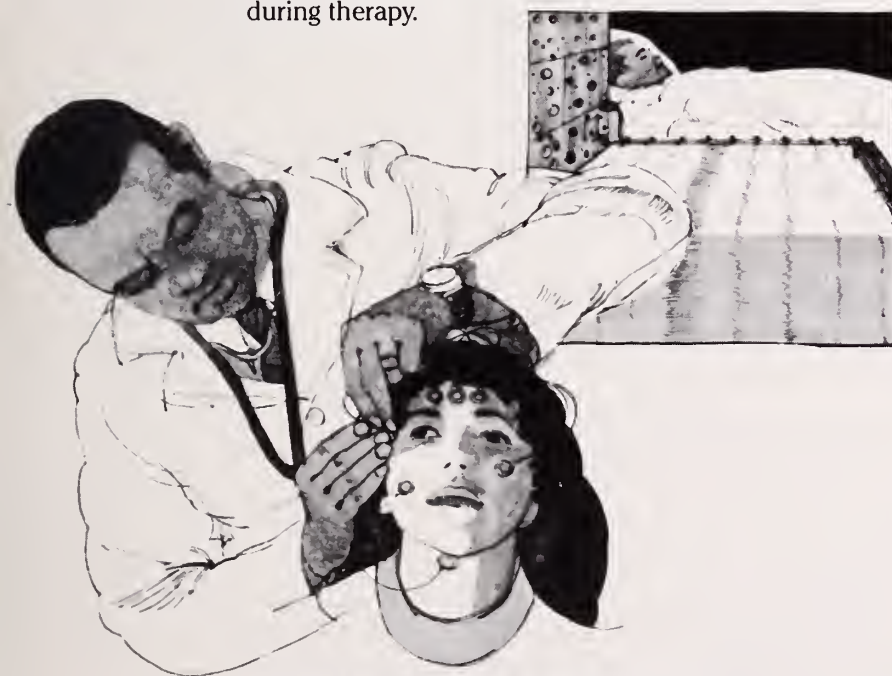
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- Contraindicated in patients who are pregnant or hypersensitive to flurazepam.
- Caution patients about drinking alcohol, driving or operating hazardous machinery during therapy.



References: 1. Kales A et al: *J Clin Pharmacol* 17:207-213, Apr 1977 and data on file, Hoffmann-La Roche Inc., Nutley, NJ. 2. Kales A: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. 3. Zimmerman AM: *Curr Ther Res* 13:18-22, Jan 1971. 4. Kales A et al: *JAMA* 241:1692-1695, Apr 20, 1979. 5. Kales A, Scharf MB, Kales JD: *Science* 201:1039-1041, Sep 15, 1978. 6. Kales A et al: *Clin Pharmacol Ther* 19:576-583, May 1976. 7. Kales A, Kales JD: *Pharmacol Physicians* 4:1-6, Sep 1970. 8. Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc* 27:541-546, Dec 1979. 9. Dement WC et al: *Behav Med* 5:25-31, Oct 1978. 10. Vogel GW: Data on file, Hoffmann-La Roche Inc., Nutley, NJ. 11. Karacan I, Williams RL, Smith JR: The

sleep laboratory in the investigation of sleep and sleep disturbances. Scientific exhibit at the 124th annual meeting of the American Psychiatric Association, Washington, DC, May 3-7, 1971. 12. Pollak CP, McGregor PA, Weitzman ED: The effects of flurazepam on daytime sleep after acute sleep-wake cycle reversal. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, June 30-July 4, 1975. 13. Data on file, Hoffmann-La Roche Inc., Nutley, NJ.

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Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, e.g., excitement, stimulation and hyperactivity.

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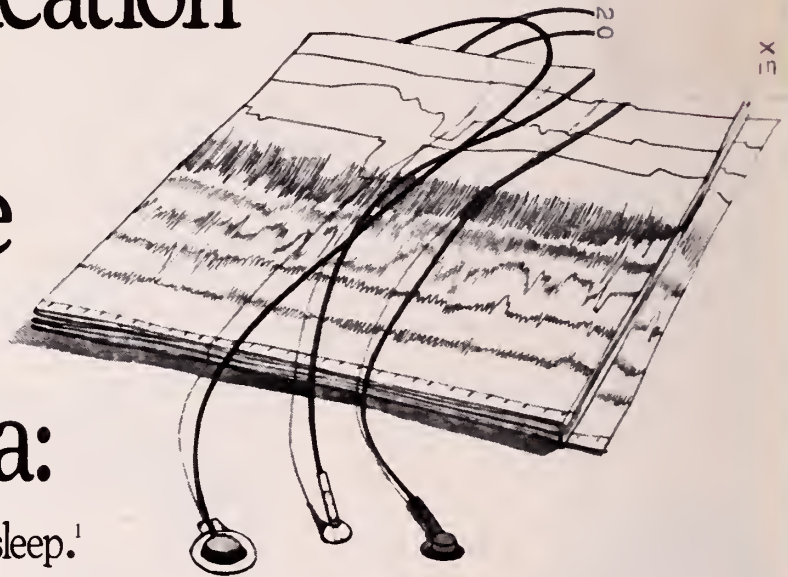
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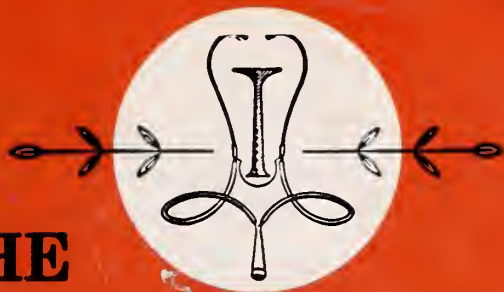
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VOL. LXXXV
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The JOURNAL of the KANSAS MEDICAL SOCIETY

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MAY 3-6

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View"**

**Congressional Candidates' Forum
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**SATURDAY
MAY 5**

*Morning
Afternoon
Evening*

**KMS First House of Delegates
KMS Reference Committee
KMS President's Installation and
Reception**

**SUNDAY
MAY 6**

Morning

KMS Second House of Delegates

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8:30-8:45	Announcements - Steve Carter Welcome - Dr. David Bouda - Chairman, Program Committee	1:10-1:30	Robert Harder, Ph.D., Secretary of SRS "Health Care - The Kansas Medicaid Program"
8:45-9:15	Carolyn K. Davis, Ph.D. Administrator - Health Care Financing Administration U.S. Department of Health and Human Services "Medicare Prospective Payment System and the Future of Physician Reimbursement"	1:30-1:50	Sylvia Hoagland - Secretary, Kansas Department of Aging "Health Care and the Kansas Elderly"
9:15-9:45	Chris Copland - Attorney at Law - Wood, Lucksinger & Epstein Congressional Fellow on Health Care with Congressman Mike Andrews "Reimbursement and its Impact on Physicians - A Federal-Legal Viewpoint"	1:50-2:10	Sister Elizabeth Stover - Chairperson, Kansas Hospital Association "Health Care - A Hospital View"
9:45-10:15	Thomas Dehn, M.D., Radiologist, Deaconess Hospital, Milwaukee, Wis. Vice-Chairman - American Medical Peer Review Association "The Future and Effects of Prospective Payment and Utilization Review"	2:10-2:30	Wayne Johnston - President, Blue Cross-Blue Shield "Health Care - An Insurance Viewpoint"
10:15-10:30	Break	2:30-2:40	Break
10:30-11:30	Panel Discussion - Questions and Answers "Impact of Prospective Payment on Physician Reimbursement and Health Care Delivery" - Moderator, Dr. Jim Gleason Panelists: Carolyn K. Davis Chris Copland Tom Dehn	2:40-3:30	Panel Discussion - Questions and Answers "Kansas Health Care Delivery - A State View" Moderator - Dr. Erwin Janssen Panelists: Robert Harder, SRS Sylvia Hoagland, Department of Aging Wayne Johnston - Blue Cross- Blue Shield Sister Elizabeth Stover - Kansas Hospital Association
		3:30	Adjournment

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**1:45 THE OTHER VIEW: THE NON-PARTICIPATING
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2:15 HEALTH CHANGES AND MARKETING

3:00 Break

**3:10 MEDICAL RECORDS UNDER PROSPECTIVE
PAYMENT SYSTEM**

**4:00 CURRENT ISSUES IN MEDICAID
REIMBURSEMENT**

**4:40 Wrapup, Evaluations, CEU Test
(as may be applicable)**

5:00 Adjournment

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Nominating Committee Report

The KMS Nominating Committee has submitted the following slate of candidates for offices for the election to be held at the annual meeting May 3-6, 1984, in Hutchinson:

PRESIDENT: F. Calvin Bigler, M.D., Garden City

PRESIDENT ELECT: Clair C. Conard, M.D., Dodge City

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Richard Meidinger, M.D., Topeka; and Roger D. Warren, M.D., Hanover

SPEAKER: G. Rex Stone, M.D., Manhattan

VICE SPEAKER: Herbert Fransen, M.D., Newton; and Edwin D. Rathbun, M.D., Liberal

AMA DELEGATE: Alex Scott, M.D., Junction City

AMA ALTERNATE: Warren E. Meyer, M.D., Wichita

AMA DELEGATE: Kermit G. Wedel, M.D., Minneapolis

AMA ALTERNATE: Linda D. Warren, M.D., Hanover

The KMS Nominating Committee encourages additional nominations, which may be presented by delegates at the annual meeting.

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This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

Contraindications: Concomitant use with other potassium-spanning agents such as spironolactone or amiloride. Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids, and during concurrent use with amphotericin B or corticosteroids or corticotropin [ACTH]). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Cumulative effects of the drug may develop in patients with impaired renal function. Thiazides should be used with caution in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease. Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic and hemolytic anemia have been reported with thiazides. Thiazides may cause manifestation of latent diabetes mellitus. The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide; dosage adjustments may be necessary. Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported. Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Anti-hypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. Triamterene has been found in renal stones in association with the other usual calculus components. Therefore, 'Dyazide' should be used with caution in patients with histories of stone formation. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide'. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased dietary intake of potassium-rich foods. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function.

Thiazides may add to or potentiate the action of other antihypertensive drugs.

Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

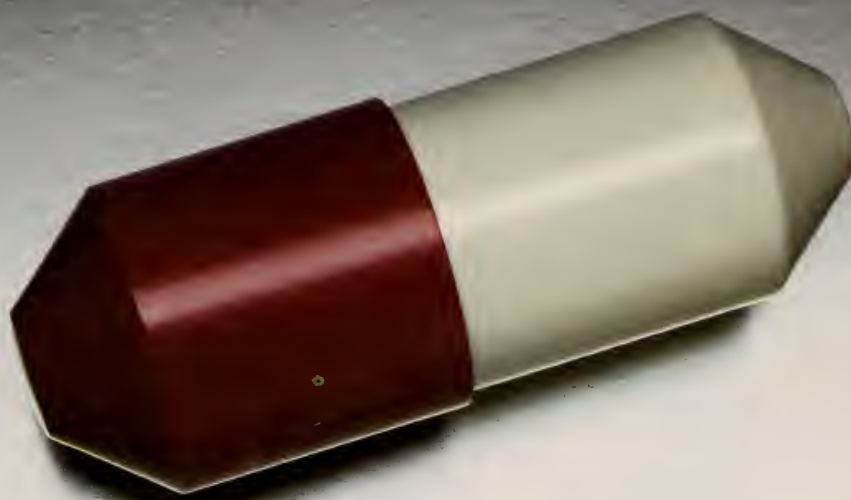
Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances; postural hypotension (may be aggravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and respiratory distress including pneumonitis and pulmonary edema, transient blurred vision, sialadenitis, and vertigo have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis have been reported. Impotence has been reported in a few patients on 'Dyazide', although a causal relationship has not been established.

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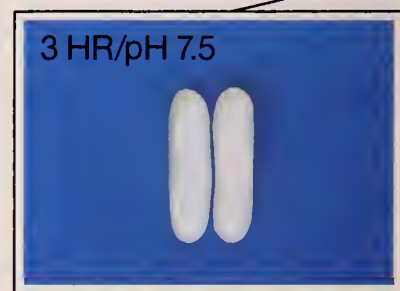
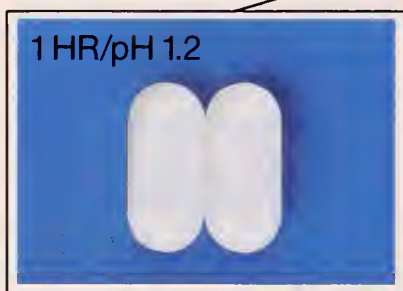


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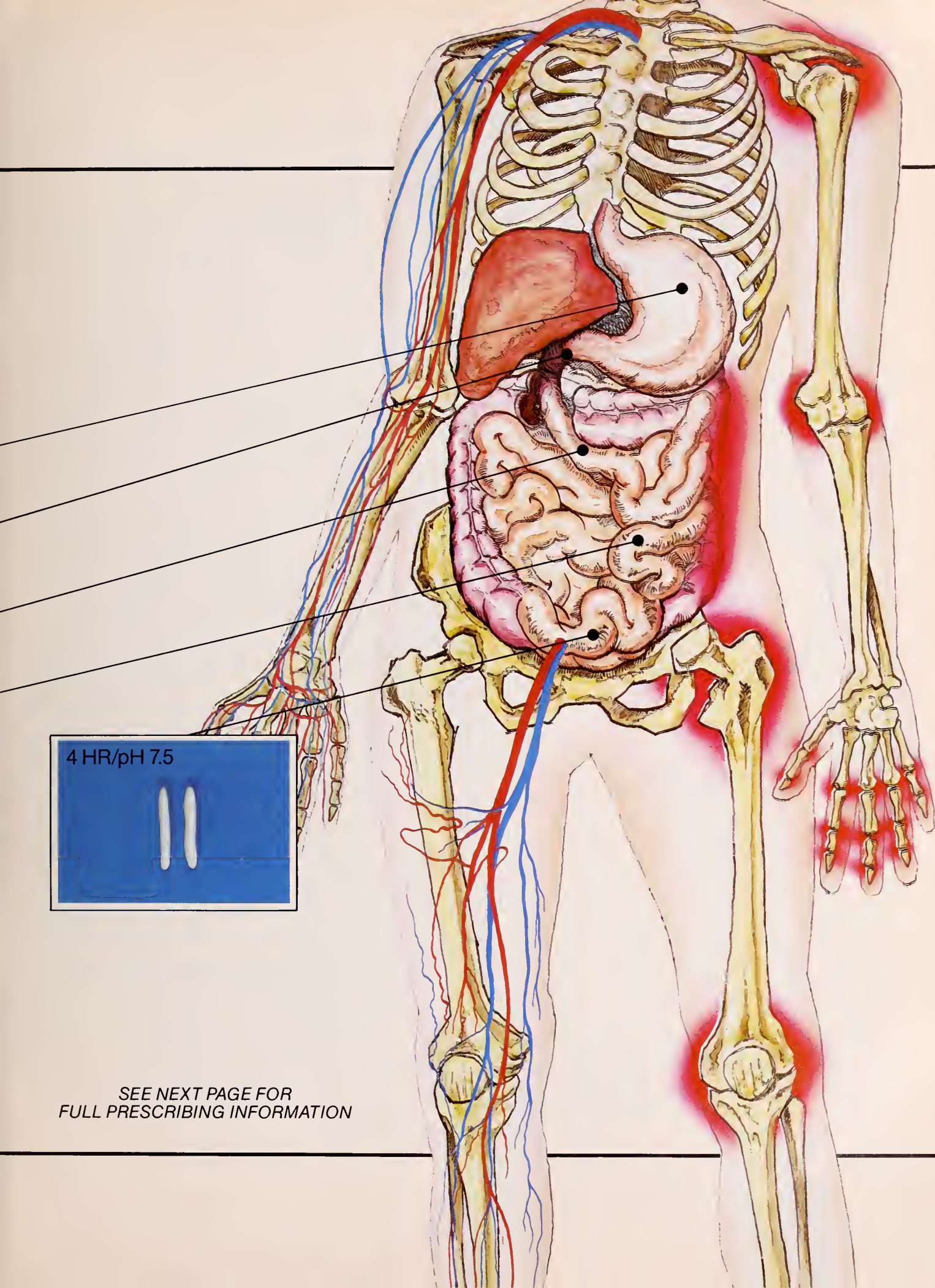
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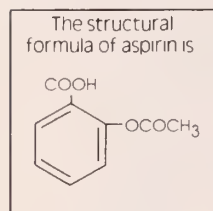


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not been established in those rheumatoid arthritis patients who are designated by the American Rheumatism Association as Functional Class IV (incapacitated, largely or wholly bedridden, or confined to wheelchair, little or no self-care). **In patients treated with Zorprin for rheumatoid arthritis and osteoarthritis, the anti-inflammatory action of Zorprin has been shown by reduction in pain, morning stiffness and disease activity as assessed by both the investigators and patients.** **In clinical studies in patients with rheumatoid arthritis and osteoarthritis, Zorprin has been shown to be comparable to conventional release aspirin in controlling the aforementioned signs and symptoms of disease activity and to be associated with a statistically significant reduction in the milder gastrointestinal side effects (see ADVERSE REACTIONS).** Zorprin may be well tolerated in some patients who have had gastrointestinal side effects with conventional release aspirin, but these patients when treated with Zorprin should be carefully followed for signs and symptoms of gastrointestinal bleeding and ulceration. **Since there have been no controlled trials to demonstrate whether or not there is any beneficial effect or harmful interaction with the use of Zorprin in conjunction with other nonsteroidal anti-inflammatory agents (NSA), the combination cannot be recommended (see Drug Interactions).** **Because of its relatively long onset of action, Zorprin is not recommended for antipyresis or for short-term analgesia.** **CONTRAINDICATIONS:** Zorprin should not be used in patients known to be hypersensitive to salicylates or in individuals with the syndrome of nasal polyps, angioedema, bronchospastic reactivity to aspirin, renal or hepatic insufficiency, hypoprothrombinemia or other bleeding disorders. Zorprin is not recommended for children under 12 years of age, it is contraindicated in all children with fever accompanied by dehydration. **WARNINGS:** Zorprin should be used with caution when anticoagulants are prescribed concurrently, since aspirin may depress platelet aggregation and increase bleeding time. Large doses of salicylates may have hypoglycemic action and enhance the effect of the oral hypoglycemics, concomitant use therefore is not recommended. However, if such use is necessary, dosage of the hypoglycemic agent must be reduced. The hypoglycemic action of the salicylates may also necessitate adjustment of the insulin requirements of diabetics. **While salicylates in large doses have a uricosuric effect, smaller amounts may reduce water excretion and increase serum uric acid.** **USE IN PREGNANCY:** Aspirin can harm the fetus when administered to pregnant women. Aspirin interferes with maternal and infant hemostasis and may lengthen the duration of pregnancy and parturition. Aspirin has produced teratogenic effects and increases the incidence of stillbirths and neonatal deaths in animals. **If this drug is used during pregnancy, or if the patient becomes pregnant while taking this drug, the patient should be apprised of the potential hazard to the fetus.** **Aspirin should not be taken during the last 3 months of pregnancy.** **PRECAUTIONS:** Appropriate precautions should be taken in prescribing Zorprin for patients who are known to be sensitive to aspirin or salicylates. Particular care should be used when prescribing this medication for patients with erosive gastritis, peptic ulcer, mild diabetes or gout. As with all salicylate drugs, caution should be exercised in prescribing Zorprin for those patients with bleeding tendencies or those on anticoagulants. **In order to avoid exacerbation of disease or adrenal insufficiency, patients who have been on prolonged corticosteroid therapy should have their therapy tapered slowly rather than discontinued abruptly when Zorprin is made a part of the treatment program.** **Patients receiving large doses of aspirin and/or prolonged therapy may develop mild salicylate intoxication (salicylism) that may be reversed by dosage reduction.** **Salicylates can produce changes in thyroid function tests.** **Salicylates should be used with caution in patients with severe hepatic damage, preexisting hypoprothrombinemia, Vitamin K deficiency and in those undergoing surgery.** **Since aspirin release from Zorprin is pH dependent, it may change in those conditions where the gastric pH has been increased as a result of antacids, gastric secretion inhibitors or surgical procedures.** **Drug Interactions:** (See **WARNINGS**) Aspirin may interfere with some anticoagulant and antidiabetic drugs. Drugs which lower serum uric acid by increasing uric acid excretion (uricosurics) may be antagonized by the concomitant use of aspirin, particularly in doses less than 2.0 grams/day. Nonsteroidal anti-inflammatory drugs may be competitively displaced from their albumin binding sites by aspirin. This effect may negate the clinical efficacy of both drugs. Also, the gastrointestinal inflammatory potential of nonsteroidal anti-inflammatory drugs may be potentiated by aspirin. The combination of alcohol and aspirin may increase the risk of gastrointestinal bleeding. **Aspirin may enhance the activity of methotrexate and increase its toxicity.** **Sodium excretion produced by spironolactone may be decreased in the presence of salicylates.** Concomitant administration of other anti-inflammatory drugs may increase the risk of gastrointestinal ulceration. Urinary alkalizers decrease aspirin's effectiveness by increasing the rate of salicylate renal excretion. Phenobarbital decreases aspirin's effectiveness by enzyme induction. **Pregnancy Category D.** See **WARNINGS** Section. **Nursing Mothers:** Salicylates have been detected in the breast milk of nursing mothers. Because of the potential for serious adverse reactions from aspirin in nursing infants, a decision should be made whether to discontinue nursing or discontinue the drug, taking into account the benefit of the drug to the mother. **ADVERSE REACTIONS: Hematologic:** Aspirin interferes with hemostasis. Patients with a history of blood coagulation defects or receiving anticoagulant drugs or with severe anemia should avoid Zorprin. Aspirin used chronically may cause a persistent iron deficiency anemia. **Gastrointestinal:** Aspirin may potentiate peptic ulcer, and cause stomach distress or heartburn. Aspirin can cause an increase in occult bleeding and in some patients massive gastrointestinal bleeding. However, the greatest release of active drug from Zorprin is designed to occur in the small intestine over a period of time. This has resulted in fewer symptomatic gastrointestinal side effects. **Allergic:** Allergic and anaphylactic reactions have been noted when hypersensitive individuals have taken aspirin. Fatal anaphylactic shock, while not common, has been reported. **Respiratory:** Aspirin intolerance, manifested by exacerbations of bronchospasm and rhinitis, may occur in patients with a history of nasal polyps, asthma, or rhinitis. The mechanism of this intolerance is unknown but may be the result of aspirin-induced shunting of prostaglandin synthesis to the lipoxygenase pathway and the liberation of leukotrienes, e.g. slow-reacting substance of anaphylaxis. **Dermatologic:** Hives, rashes, and angioedema may occur, especially in patients suffering from chronic urticaria. **Central Nervous System:** Taken in overdoses, aspirin provides stimulation which may be manifested by tinnitus. Following initial stimulation, depression of the central nervous system may be noted. **Renal:** Aspirin rarely may aggravate chronic kidney disease. **Hepatic:** High doses of aspirin have been reported to produce reversible hepatic dysfunction. **OVERDOSAGE:** Overdosage, if it occurs, would produce the usual symptoms of salicylism: tinnitus, vertigo, headache, confusion, drowsiness, sweating, hyperventilation, vomiting or diarrhea. Plasma salicylate levels in adults may range from 50 to 80 mg/dl in the mildly intoxicated patient to 110 to 160 mg/dl in the severely intoxicated patient. An arterial blood pH of 7.1 may indicate serious poisoning. The clearance of salicylates in children is much slower than adults and should receive due consideration when aspirin overdoses occur in infants, salicylate half-lives of 30 hours have been reported in infants 4-8 months old. Treatment for mild intoxication should include emptying the stomach with an emetic, or gastric lavage with 5% sodium bicarbonate. Individuals suffering from severe intoxication should, in addition, have forced diuresis by intravenous infusions of sodium bicarbonate and dextrose or sodium lactate. In extreme cases, hemodialysis or peritoneal dialysis may be required. **(A plasma salicylate level of 160 mg/dl in an adult is usually considered lethal.)** **DOSAGE & ADMINISTRATION:** *In order to achieve a zero-order release, the tablets of Zorprin should be swallowed intact.* **Breaking the tablets or disrupting the structure will alter the release profile of the drug.** **It is recommended that Zorprin be taken with sufficient quantities of fluids (8 oz. or more).** **Adult Dosage:** For mild to moderate pain associated with rheumatoid arthritis and osteoarthritis, the recommended initial dose of Zorprin is 1600 mg (2-800 mg tablets) twice a day. Because of Zorprin's prolonged release of aspirin into the bloodstream, Zorprin tablets may be taken as a b.i.d. dose. Further adjustment of the dosage should be determined by the physician, based upon the patient's response and needs. Since it will take 4-6 days to reach steady-state levels of salicylic acid with Zorprin, it is recommended dosages be given for at least one week before further adjustment. In general, patients with rheumatoid arthritis seem to require higher doses of Zorprin than do patients with osteoarthritis. **Zorprin is not recommended for children below the age of 12.** **HOW SUPPLIED: Zorprin Tablets 800 mg;** plain, white capsule-shaped tablets. **Bottles of 100 Tablets — NDC 0524-0057-01.** **Caution:** Federal law prohibits dispensing without prescription. **U.S. Patent No. 4,308,251.** **Manufactured and Distributed by: BOOTS PHARMACEUTICALS, INC., Shreveport, Louisiana 71106 U.S.A.**

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A U X I L I A R Y N E W S

An Open Letter to Kansas Physicians

This is an update on the Kansas Medical Society Auxiliary and, in particular, activities during my year as President. The Auxiliary functions as a support group to the Kansas Medical Society. In this capacity, the Auxiliary helped implement the "Forums on Aging," a project originating with the Medical Society. Under the direction and assistance of Val Braun of the KMS office, six county auxiliaries will have staged forums during this past Auxiliary year. These counties with their chairmen are: Barton County, Sue Alderson (Tom); Douglas County, Ruth Mitchell (Alex); Flint Hills, Vickie Ryan (Scott); Sedgwick County, Suzie Ahlstrand (Richard); Shawnee County, Katie Pyle (Lucien);

and Saline County, Martha Brummett (Richard). Others are in the planning stages.

While the forums held in the various counties were each unique entities, they had some features in common. Each had a local physician and legislator as guest speakers, and all had the following government agencies as co-sponsors: Kansas Department of Aging, Kansas Insurance Department, Kansas Department of Health and Environment, and Medicare. I wish to publicly thank all of these participating agencies. They did a great job.

The rest of the Auxiliary's activities have been just as notable. Membership remains strong under the chairmanship of Betty Glover (Richard). Auxiliaries across the state realize that the only way medicine can exist is for all of us — physicians and spouses alike — to work together for a common goal. Singly, we could be blown away like the Kansas topsoil during the dust bowl days. (Does this date me?)

The American Medical Association Education and Research Fund (AMAERF) has also done very well this year under the directorship of Nancy Macy (Ted). AMAERF funds assist with the schooling of students, interns and residents, and support important scientific and medical research. In addition to direct gifts and memorials, there have been numerous money generating programs and projects.

The big money raiser finale of the year for AMAERF will come with the Casino Party at the State Convention, Friday night, May 4, hosted by the Reno County Auxiliary in Hutchinson. It is going to be a fantastic evening of fun and games. Be sure to send in your reservations early since we are expecting a huge crowd. You will miss the Party of the Year if you don't come! Besides, you will be supporting a very worthy cause.

I also want to stress that we need and solicit your spouse's participation in the State Auxiliary Convention meetings in Hutchinson, May 3-5. Any organization can remain strong only as long as all of its members continue to be active and alert to the needs of the entire membership.

Thank you individually and collectively as the KMS, for your support this past year. I have enjoyed myself immensely and feel fortunate to represent such an energetic and truly concerned group as the Kansas Medical Society Auxiliary.

Diane Sanders

President

Kansas Medical Society Auxiliary

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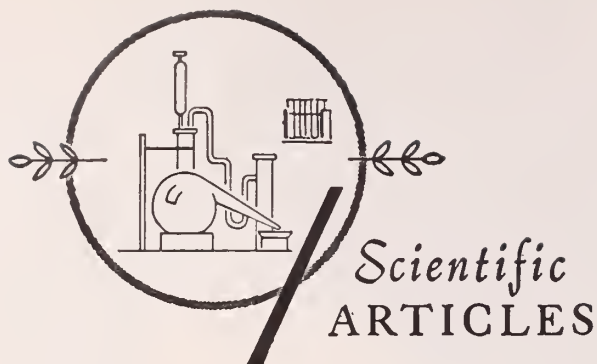
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Anesthesia in Carotid Endarterectomy

LARRY O'HOLLERAN, M.D.;* JAMES E. FRENCH, M.D.† and
S. JIM FARHA, M.D.,‡ *Wichita*

VARYING OPINIONS regarding the choice for anesthesia for carotid endarterectomy appear in the literature.¹⁻³ Between 1975 and 1980, regional anesthesia with selective shunting was utilized at our institution. Since 1980, more liberal use has been made of general anesthesia with routine shunting. This retrospective study compares the two groups of patients in terms of morbidity, mortality, and postoperative hospital stay.

Materials and Methods

From 1975 through 1982, 411 carotid endarterectomies were performed on 363 patients at this institution by one vascular surgical team; 272 were performed under regional anesthesia with selective shunting, and 139 endarterectomies were performed under general anesthesia with routine indwelling shunts. Patient ages ranged from 52 to 88 years for males with an average of 65.2 years; and 33 through 79 years for females with an average of 63.8 years. The majority of these patients presented with hypertension or arteriosclerotic heart disease.

All patients underwent arch and 4-vessel biplane angiography. Indications for arteriography were: (1)

cerebral transient ischemia attacks, 296 or 82 per cent; (2) previous completed strokes with continued cerebral transient ischemic attacks, 35 or 9.2 per cent; and (3) asymptomatic bruits, 32 or 8.8 per cent. No significant differences were noted in the clinical picture of either group. Regional anesthesia was by cervical block with 1 per cent xylocaine performed by an anesthesiologist, and vital signs were continuously monitored by a nurse anesthetist. Premedication was not routine. Monitoring by continuous electrocardiogram and radial artery pressure line was maintained and oxygen was delivered by mask at 40 per cent concentration.

Meticulous care to minimize manipulation of the vessels as isolation tapes were placed followed the details described by Spencer⁴ and Rainer.⁵ The carotid sinus was infiltrated with 1 per cent xylocaine and systemic heparinization was performed. If awake, the patient was monitored for such indications as consciousness, slurred speech, visual disturbances. An internal shunt was inserted if signs of cerebral ischemia developed but omitted if cross-clamping of the carotid arteries was tolerated. In the general anesthetic group, systemic heparinization, continuous EKG and radial line monitoring, and indwelling shunts were routinely utilized.

All precautionary measures were taken during the procedure as well as following closure of the artery to prevent cerebrovascular accidents. Postoperatively, all patients were carefully monitored in the Surgical Intensive Care Unit for 12-24 hours. On the first postoperative day, any drains remaining were

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removed and patients without significant problems were transferred to floor care.

Results

The peri-operative mortality rate in these patients was 0 per cent for patients undergoing general anesthesia and 1.1 per cent (3 patients) for patients undergoing regional anesthesia. Two of the deaths were secondary to postoperative myocardial infarction and one death occurred following a severe postoperative hypertensive episode with subsequent shock.

Permanent neurological deficits occurred in 11 patients (2.6%), four (2.9%) under general anesthesia, and seven (2.5%) under regional anesthesia. Exact timing and etiology of stroke was not determined for those patients who underwent general anesthesia. However, for the seven patients who underwent regional anesthesia, two strokes occurred during mobilization of the carotid artery and five occurred during the immediate postoperative period.

Other complications encountered included six wound hematomas requiring reoperation (all but one of these occurring prior to the routine use of 24 hour closed suction drainage); two cardiac arrhythmias; one cardiac arrest; two wound infections; and one painful scar requiring revision. The breakdown between the two groups is shown in *Table I*.

Of the 272 endarterectomies performed under regional anesthesia, shunts were deemed necessary in 23 cases or 8.4 per cent.

Postoperative hospitalization time ranged from two to 24 days for general anesthesia and two to 23 days for regional anesthesia patients. If those patients who subsequently underwent additional cardiovascular procedures were eliminated, the average hospital stay was 2.9 days for general and 3.2 days for regional anesthesia.

Discussion

Controversy prevails regarding the ideal anesthetic technique for carotid endarterectomy with proponents of general and regional techniques showing excellent results with their respective methods.^{1-3, 7} The crux is the selection of patients needing particular protection of the cerebral circulation and the method of achieving it.^{1, 2, 6} Carotid stump pressures are advocated to avoid routine shunting under general anesthesia,⁸ but this technique remains controversial. A review of 290 patients undergoing the operation disclaimed its value and advocated local anesthesia with intraoperative assessment of motor ability and consciousness as the only absolute criteria of need for shunting. Proponents of surgery under general anesthesia counter with claims for its greater ease of performance and better cerebral protection.^{1, 9}

Shortcomings in assessing adequate collateral circulation lead some to advocate routine shunting in partially occlusive lesions. Avoidance of routine shunting of all general anesthesia patients requires selection of patients who can tolerate carotid cross-clamping without neurologic damage. Some report success with EEG monitoring, but we have had no experience with this so far. Alternative techniques such as visualization of back bleeding and monitoring jugular oxygen saturation are criticized^{2, 8} as unreliable, and at this time it remains for each surgeon to devise overall management to assure acceptable results.¹

Advantages of shunt placement lie not only in the continuous cerebral protection afforded the patient but also in allowing for a well controlled, unhurried operation that may be crucial in cases of complicated lesions.^{1, 10, 11} Although critics of routine shunting cite the disadvantage of tubing in the operative

(Continued on page 121)

TABLE I
COMPARISON OF COMPLICATIONS WITH GENERAL AND REGIONAL ANESTHESIA

	<i>Postoperative Complications Total</i>	<i>General Anesthesia</i>	<i>Regional Anesthesia</i>
Mortality	3 (0.7%)	0 (0%)	3 (1.1%)
Stroke	11 (2.6%)	4 (2.9%)	7 (2.5%)
Cardiac Arrest	1 (0.2%)	1 (0.7%)	0 (0%)
Cardiac Arrhythmia	2 (0.5%)	1 (0.7%)	1 (0.3%)
Wound Hematoma	6 (1.4%)	2 (1.4%)	4 (1.4%)
Wound Infection	2 (0.5%)	0 (0%)	2 (0.7%)
Painful Scar	1 (0.2%)	0 (0%)	1 (0.3%)

Hormonal Patterns in Menstrual Dysfunction

PAUL REITH, M.D. and RAYMOND SCHWEGLER, M.D., Ph.D., *Lawrence*

THIRTY COLLEGE women with a history of menstrual dysfunction were evaluated for hormonal patterns at Watkins Memorial Hospital, Lawrence. To expedite the evaluation, a simplified sequence was utilized: (1) a urinary pregnancy test; (2) an oral progesterone withdrawal test; and (3) a blood test for luteinizing hormone (LH), follicle stimulating hormone (FSH), prolactin (PRL), a total testosterone (TT), and a thyroxine (T4) level or a thyroid stimulating hormone (TSH).

The above sequence was initiated to more rapidly evaluate the many women who present with menstrual dysfunction at the student health facility, and who often also request the oral contraceptive pill. Because it is well known that women with prior oligomenorrhea and amenorrhea are more likely to develop postpill amenorrhea, it is prudent to identify those who might be more prone to prolonged anovulation and infertility following the use of oral contraceptive steroids.¹

Kletzky *et al.*'s classification of secondary amenorrhea based on distinct hormonal patterns was used. This classification included: a "polycystic ovary" group with an elevated basal and widely fluctuating LH, low FSH, and a normal estradiol (E_2); "hypothalamic-pituitary dysfunction" with a normal baseline LH, FSH, and E_2 and a fluctuating LH; "hypothalamic-pituitary failure" with a normal or low basal LH and FSH, a low E_2 with minimal or absent fluctuation of LH; and "ovarian failure" with a high baseline LH and FSH and a low E_2 .²

Kletzky *et al.*'s protocol was modified with substitution of the oral progesterone withdrawal test for serum E_2 to reduce costs, as it is known that a positive withdrawal flow following oral or intramuscular progesterone indicates a serum E_2 of at least 50 pg/ml.³ This substantiates follicular maturation and intactness of the hypothalamic-pituitary axes. A TT test was added as a measure of androgen and also to rule out adrenal or ovarian neoplasms which would be expected to result in levels of TT greater than 250 ng/ml. PRL was added to screen for hyperprolactin states, as Kletzky *et al.* included patients with pituitary tumors in their "hypothalamic-

pituitary failure" group. The classification then included the following categories: A "polycystic ovary (PO)" pattern was defined by a TT level greater than 95 mg/ml; an LH/FSH ratio greater than 3.0; or an absolute LH level greater than 30 mlu/ml. The "hypothalamic-pituitary dysfunction (HP-Dys)" group was characterized by normal or low baseline gonadotropins. A "hyperprolactin (HPRL)" group included a smaller number of patients with a more marked elevation of PRL, an inverted LH/FSH ratio, a normal or increased TT, and evidence of an adenoma on tomography of the pituitary sella. A unique subgroup of hyperprolactinemic patients had only mild elevation of PRL, increased LH/FSH ratio, normal or increased TT, and no evidence of pituitary adenoma on lateral skull film tomography. This was termed a mixed "hyperprolactin-polycystic ovary (HPRL-PO)" pattern. One woman with an elevated FSH was said to have "ovarian failure (OF)."

Methods

Thirty college women were evaluated during a two-year period, some with oligomenorrhea or menstrual cycles with 35-50 days between flows, or amenorrhea with absent menstruation for four months or more. The sequence of tests began with the urinary pregnancy test, followed by an oral progesterone withdrawal test in those with delayed or absent periods, and a morning blood test five to eight days after onset of menses or withdrawal flow.

The Neocept kit was used for determination of urinary HCG. The patient was instructed to avoid fluids for ten hours overnight and to collect an early morning specimen in an inert container. A specific gravity of 1.020 was required. This test is quite sensitive, measuring the beta-subunit of HCG in a concentration to only 0.15 units.

Medroxyprogesterone (Provera), 10 mgm/day, was administered orally for five days for the oral progesterone withdrawal test. An onset of menstrual flow within five days was termed a positive result, although ten days were sometimes required before a flow was reported.

Serum was drawn between 9 and 10 AM after a ten-hour overnight fast. Three tubes were drawn 15 minutes apart, and the serum pooled to give an average value. Included were: LH, FSH, TT, PRL, and either a T₄ or TSH. Prolactin and all assays were

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done in 14 women; selected tests were done in the other 16 subjects. All radioimmunoassays were sent to BioScience Labs. Normal values for hormones tested included: LH, 6-30 mIU/ml; FSH, 4-30 mIU/ml; TT, 30-95 ng/dl; PRL, 6-22 ng/ml; T_4 , 5-12 mcg/dl; and TSH, 1.9-5.4 IU/ml.

Results

A PO pattern was found in 13 women (43%) with a TT level greater than 95 ng/ml, an LH/FSH ratio greater than 3.0, or an absolute LH greater than 30 mIU/ml, with a normal serum PRL. The mean TT was 99.9 ± 40 ng/ml, mean LH/FSH ratio 4.5 ± 2.9 , and mean LH 29 ± 13 mIU/ml. Laparoscopy disclosed polycystic ovaries in three of the above patients who also had abnormal findings on pelvic examination.

The hyperprolactin group was composed of eight patients (27%), two with higher PRL levels (130 ng/ml and 42 ng/ml), an LH/FSH ratio less than 1.0, a TT in one of 114 ng/dl, and evidence in both of a microadenoma on hypocyndial tomography of the sella. The other six with a mixed HPRL-PO pattern had only mildly elevated mean PRL levels of 30.7 ± 9 , an increased LH/FSH ratio of 4.5 ± 3.7 , and a high normal level of TT, 72.5 ± 22 ng/ml. Tomography of the sella was read as normal in two of these patients, and a thyrotrophic releasing factor (TRF) test showed an exaggerated response of PRL in the same two. Thyroxine levels and baseline TSH were normal in all six.

Eight (27%) had HP-Dys patterns with normal baseline hormonal values. The mean LH was 14.8 ± 5.5 mIU/ml, mean FSH 9.0 ± 1.2 mIU/ml, LH/FSH ratio 1.9 ± 0.8 , mean TT 63 ± 29.9 ng/ml,

mean PRL 7.3 ± 2.3 ng/ml, T_4 7.6 ± 1.3 mcg/dl, and mean TSH 3.1 ± 1.4 IU/ml. One 46-year-old student had an increased FSH of 46 mIU/ml, and was thought menopausal. None of the 30 were chemically hypothyroid. None had HP-failure with very low gonadotropins.

The OPWT was 85-100 per cent positive in the PO group, 100 per cent positive in the HPRL-PO subgroup, negative in the HPRL group with pituitary adenomas, and variably positive, 42-71 per cent, in the HP-Dys group. Those in the PO group were greatest in mean weight, $+18 \pm 23\%$ above ideal; the HPRL patients were $+13 \pm 13\%$ above ideal; the HPRL-PO group also $+12 \pm 14\%$ above ideal and the women with HP-Dys were thinner, $+8 \pm 7\%$.

Table I displays the means and standard deviation of the PO, HPLR, HPRL-PO, and HP-Dys groups.

Discussion

The above sequence of tests and hormonal assays helped to categorize our patients into distinct groups.

Considering specific tests in the PO pattern, the LH was greater than 30 mIU/ml in 38 per cent and greater than 29 mIU/ml in 69 per cent of patients; the TT was greater than 95 ng/ml in 57 per cent and greater than 90 ng/ml in 71 per cent; and the LH/FSH ratio was greater than 3.0 in 70 per cent. At least one of the above tests met the criteria for polycystic ovary syndrome in 86 per cent. Of the two patients remaining, one had an elevated androstenedione; her clinical hyperandrogen status was not reflected in the TT test. The other individual most likely had oligomenorrhea associated with obesity; her clinical hyperandrogen status also was not documented with

TABLE I
COMPARISON OF THE GROUPS (MEAN AND STANDARD DEVIATION)

	OPWT	LH	FSH	LH/FSH	T	PRL	T_4	TSH	Weight
PO	85-100%	29.0 ± 12.8 (13)	7.3 ± 0.9 (10)	4.5 ± 2.9 (10)	99.9 ± 40.4 (14)	9.6 ± 1.6 (12)	8.7 ± 1.4 (7)	3.1 ± 0.8 (4)	+18% ± 23 (4)
HPRL	0%	8.5 ± 9.2 (2)	11.0 ± 5.7 (2)	0.7 ± 0.4 (2)	114.0 (1)	86.0 ± 60.2 (2)	5.5 ± 1.3 (2)	3.8 ± 1.3 (2)	+13% ± 13 (2)
H-PRL+PO	100%	30.7 ± 15.2 (6)	8.0 ± 2.2 (6)	4.5 ± 3.7 (6)	72.5 ± 21.6 (6)	30.7 ± 9.0 (6)	6.5 ± 0.8 (5)	3.5 ± 0.9 (4)	+12% $\pm 14\%$ (6)
HP-Dys	42-71%	14.8 ± 5.5 (6)	9.0 ± 1.2 (5)	1.9 ± 0.8 (4)	63.0 ± 29.9 (4)	7.3 ± 2.3 (7)	7.6 ± 1.3 (6)	3.1 ± 1.4 (4)	+8% ± 7 (7)

the TT alone.

One woman with findings of ovarian enlargement on ultrasound of the pelvis and also characteristic Stein-Leventhal ovaries on laparoscopy had a low LH of 9 mIU/ml. Interestingly, the LH did not rise to a more normal adult level with weight gain from 20 per cent below ideal to normal weight. The patient was emotionally depressed but was not taking antidepressant medication. The low LH may have reflected hypothalamic dysfunction, a pattern similar to that seen in anorexia nervosa, except for the much higher TT and increased LH/FSH ratio. Givens *et al.* have commented on a "high LH" and "a normal LH" pattern in polycystic ovary syndrome, but an explanation for the variant LH pattern is not yet clear.⁴ No straight line correlation was found between body weight/ideal weight and LH in this series.

Several women with a PO pattern had "equivocal positive" urinary pregnancy tests with the Neocept kit for urinary HCG. Their higher urinary LH most likely cross-reacted with the antibody for HCG, as the kit is not highly selective for beta-subunit HCG. False positive tests for urinary HCG in postmenopausal women with elevated gonadotropins have occurred.

The FSH was increased in the patient with menopausal symptoms. Elevation of her FSH greater than 40 mIU/ml made the possibility of fertility small.⁵ Nevertheless, a few women with premature ovarian failure may actually have a "resistant ovary syndrome." Laparotomy and ovarian biopsy is recommended in these women with secondary amenorrhea and infertility but with a normal karyotype, as ovulation can sometimes occur after discontinuing suppression with exogenous estrogen, or with gonadotropin stimulation.⁶

The LH/FSH ratio was increased in both the polycystic ovary syndrome and the HPRL-PO patterns, and decreased in the HPRL pattern with a pituitary adenoma. A reversal of the LH/FSH ratio was noted in women athletes with amenorrhea; they revert to an earlier developmental pattern with a reversed LH/FSH ratio like that seen in early puberty.

Interestingly, the TT level was greater than 95 ng/ml in more than one-half of the women with the PO pattern and was high normal in the HPRL-PO group. "Free" or unbound testosterone was not measured, but this measurement has been helpful in identifying women with mild hyperandrogen status.⁷ In a select few women, dihydroepiandrosteredione (DHEA-S) levels were checked for evidence of an adrenal component. Kirschner's studies have shown that the ovary is the source of excess

androgen in 90 per cent of hyperandrogen women; the adrenal gland is the source in only 10 per cent.⁸ The oral contraceptive pill not only suppresses the ovary via LH, but also suppresses the adrenal via ACTH. Conversely, suppression of ACTH with corticosteroid also diminishes ovarian hormone production.⁹ If virilization is noted, a 17-hydroxyprogesterone (17-OH-P) level is added, as 21-hydroxylase deficiency may be present during adulthood without short stature.¹⁰

A prolactinoma is more likely if the serum PRL is greater, a mentioned cutoff point being 50 ng/ml.¹¹ One student with a PRL of 42 ng/ml had a blunted response to thyroid releasing factor (TRF), the PRL drawn prior to and 30 minutes after an intravenous bolus of 500 mcg protirelin. A TRF test in two of the students with a mixed HPRL-PO pattern demonstrated an exaggerated PRL response, suggesting increased PRL reserve. Corenblum and Taylor reported a similar exaggerated response of PRL to TRF in women with the polycystic ovary syndrome who had elevated baseline PRLs; they thought this compatible with lactotroph hyperplasia.¹² None of the women were chemically hypothyroid. Normal T₄ and TSH levels were found in all, ruling out subclinical hypothyroidism. It has been common practice to give thyroid hormone to women with oligomenorrhea and infertility. It has been suggested that augmented thyroid hormone levels might influence the menstrual cycle by altering the metabolism of estrogens, perhaps influencing hypothalamic or pituitary function as well.

In general, the full battery of tests including the urinary pregnancy test followed by an oral progesterone withdrawal test, and subsequently by the pooled serum sample for LH, FSH, PRL, and TT has been found to be helpful for more rapid screening. Tests for "free" androgen, TT, and also DHEA-S have recently been included for the woman who is hirsute. With signs suggestive of virilization, a 17-OH-P test is done to rule out an adult 21-hydroxylase deficiency. If galactorrhea is noted on physical examination, a PRL as well as an LH and FSH is indicated, and a TSH is added especially in the presence of a palpable goiter. Further tests for androgens are usually done in women with galactorrhea or in followup studies of those with hyperprolactinemia, as androgens (especially adrenal androgen), DHEA-S, and also free testosterone will be elevated.¹³ Women with more markedly elevated PRLs and an inverted LH/FSH ratio are more thoroughly evaluated with tomography of the sella or CAT scan, and also TRF testing for PRL reserve. If

(Continued on page 123)

Post NICU Development

W. JUNE HOLSTRUM, Ph.D., *Wichita*

REGIONALIZED neonatal intensive care centers have significantly improved the care of premature and sick infants. The increasing number of infants who survive complications during the neonatal period has led to questions regarding the developmental status of these infants. This study was undertaken to determine the developmental status of neonatal intensive care unit (NICU) graduates who were born in 1981 at the Regional Perinatal Care Center at Wesley Medical Center in Wichita, and who participated in the followup program.

The followup clinic was established to evaluate the progress of transported infants or infants of transported mothers. In 1981, 897 babies were admitted to the NICU and 789 survived (mortality rate = 12%). The data analyses for this study are based on 163 of these infants who were subsequently evaluated in clinic. Most (120) were transported infants; nine were local inborn infants; 34 were maternal transports.

Age at Testing

Children are routinely seen in clinic at 3-4 months and 8-9 months (age adjusted for prematurity). Children with questionable or abnormal development were seen more often and for a longer period of time. Ages at final visit ranged from 3 to 18 months. Fifty-nine children were seen only once, and 30 were still being seen past 9 months of age.

Developmental Status

At each clinic visit the children were evaluated by

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a physical or occupational therapist, a developmental psychologist, and a neonatologist. Three measures of developmental status were used — mental, motor, and neurological — and each child was given a developmental rating (DR) of 1, 2, or 3 for each area: 1 = normal; 2 = questionable/mildly abnormal; 3 = moderately/severely abnormal.

Ratings for mental and motor development were assigned by the psychologist. Children who were at least 5 months old were assessed by Bayley Scales of Infant Development.¹ Developmental quotients (DQs) in the normal range were given a DR of 1; quotients one standard deviation (SD) below normal were given a DR of 2, and quotients two SDs below normal were given a DR of 3.

The DR assigned for neurological development was based on the presence or absence of abnormal tone, reflexes, muscle strength, and movement. Neurological status was evaluated by the therapist and the neonatologist.

Developmental Outcome

One hundred nineteen infants (73%) were judged to be within normal limits (WNL) in mental and neurological development. One hundred fourteen (70%) were WNL in motor development. Approximately 20 per cent of the infants evaluated had questionable or mildly abnormal development, and approximately 10 per cent were clearly abnormal.

Variables

Two analyses were run. The first was a series of chi-squares to define association with bleeds, seizures, or congenital anomalies. Each condition was rendered as present or absent. For each analysis, two

TABLE I
TEST FOR SIGNIFICANT RELATIONSHIP BETWEEN DEVELOPMENTAL OUTCOME AND BLEEDS
CONGENITAL ANOMALIES OR SEIZURES

Variables	Mental		Motor		Neurological	
	F*	p†	F	p	F	p
Bleeds	5.005	.0246	13.855	.0002*	4.444	.0350
Congenital anomalies	5.986	.0144	6.085	.0136	1.471	.2252
Seizures	12.681	.0004*	18.059	.0001*	4.812	.0283

levels of developmental rating were used: 1 = normal; 2 = not normal.

Poor outcome for mental development was associated with seizures. Delayed motor development was associated with bleeds and seizures. Abnormalities in neurological development were not significantly related to any of the three variables tested. The presence of congenital anomalies was associated with less optimal mental and motor outcome, but that association did not reach significance (*Table I*).

A multivariate analysis was used to test for significant associations of continuous variables: birthweight, gestational age, Apgar at one minute, Apgar at five minutes, and respiratory status (measured by total number of days respiratory assistance was needed). Only the respiratory status was associated with poor mental development; the longer a child needed respiratory assistance, the less optimal was mental development.

Abnormal neurological outcome was associated with birth weight and respiratory status. The smaller the infant and the more respiratory assistance needed, the more likely s/he was to have neurological impairment (*Table II*).

Discussion

These results are consistent with other followup studies.²⁻⁴ Approximately 30 per cent of the NICU graduates in this study had some impairment. Infants who experience intensive care are at greater risk for developmental disabilities, yet only 38 per cent of NICU survivors were invited back for followup, and only 21 per cent of the total were actually seen. If the purpose of followup is early identification and intervention of handicapped children, then many infants are being missed.

Long term followup studies indicate that NICU graduates are also at risk for attention deficits,⁵ behavioral disorders,⁶ poor social skills,⁷ and slower information processing.^{8, 9} These problems may reflect the more subtle insults sustained during the neonatal period and are highly associated with lack of success in school.¹⁰ The effectiveness of early intervention programs for children with developmental disabilities is clearly documented.^{11, 12} Therefore, it is important to follow these high risk children and begin early intervention programs for those with identified developmental delays.

References are available from Dr. Holstrum, UKSM-Wichita, 1010 No. Kansas, Wichita KS 67214.

TABLE II
TEST FOR SIGNIFICANT RELATIONSHIP BETWEEN DEVELOPMENTAL OUTCOME AND BIRTHWEIGHT, GESTATIONAL AGE, 1 AND 5 MINUTE APGAR SCORES AND RESPIRATORY STATUS

Variables	Mental		Motor		Neurological	
	F*	p†	F	p	F	p
Birthweight	.52	.4736	.41	.5252	9.50	.0024*
Gestational age	.03	.8607	.18	.6717	4.45	.0366
Apgar 1 min	3.93	.0492	2.41	.1229	3.04	.0834
Apgar 5 min	1.54	.2164	.53	.4678	1.70	.1939
Respiratory distress	6.92	.0094*	4.69	.0322	13.19	.0004*

The Many Faces of Mono

HENRY JOHNSTON, M.D.* and MARTIN J. RUBINOWITZ, M.D.,† Denver, Colorado

INFECTIOUS mononucleosis (IM) can be a protean disease. Although most cases present typically with sore throat, fever, and atypical lymphocytosis and have a short course, there are many exceptions. Rarely, serious complications may occur.¹ In order to avoid disastrous treatment errors, the clinician must be aware of life threatening diseases that may be mimicked by IM.

The following case illustrates some of these problems.

Case One

A 39-year-old white female was admitted to Saint Joseph Hospital in Denver on November 6, 1981. She had a one-week history of fever up to 38.9°C, chills, achiness, and fatigue. Two days prior to admission she developed four-extremity purpura which was initially patchy and then became confluent. The results of her physical examination were normal except for the extensive purpura (*Figure 1*).

Abnormal laboratory findings included serial hematocrits of 36, 27, and 33; white blood counts of 9,700 and 14,500 with 27-49% lymphocytes with many atypical forms reported; platelet counts of 156,000, 97,000, and 359,000; reticulocyte counts of 5.8 and 9.3%; an elevated erythrocyte sedimentation rate of 130; elevated LDH (720), alkaline phosphatase (220) and SGOT (168); + direct Coombs test, cryoglobulins and cold agglutinins; low normal fibrinogen of 200; and elevated fibrin split products of 320. Studies that yielded normal results included an ANA, urinalysis, blood, throat and urine cultures, and chest x-ray. The monospot, heterophile (1:160 guinea pig titer) and Epstein-Barr virus (EBV) titer (1:320) were all positive.

Diagnoses considered at the onset were meningococcal septicemia, Rocky Mountain spotted fever, and disseminated intravascular coagulation. The patient was treated for these illnesses with heparin briefly and a ten-day course of chloram-

phenicol and penicillin. On November 10, the patient had a skin biopsy which showed necrotizing vasculitis consistent with periarteritis nodosa. This diagnosis was essentially excluded by normal vertebral, carotid, renal, celiac, and mesenteric arteriography. Prednisone therapy, 80 mg/day was instituted promptly. Her purpuric rash evolved like a burn with scarring and contractures of the skin. The rest of her symptoms resolved and she was discharged on November 24.

Subsequently, her liver functions and sedimentation rate normalized. Her skin lesions required grafting following which she was tapered off prednisone.

Comment

Although this patient had many of the reported characteristics of IM such as atypical lymphocytosis, positive monospot, heterophile and EBV titers, hemolytic anemia, thrombocytopenia, cryoglobulinemia, cold agglutininemia and hepatic dysfunction, her skin manifestations may be unique. Schumacher and Barclay² reported toxic vascular purpura in 6.9 per cent of their 450 cases. Bleeding manifestations did not necessarily correlate with the degree of thrombocytopenia and included palatal and gingival bleeding, hemoptysis, epistaxis, hematuria, gastrointestinal bleeding, purpura, and mild dermal vasculitis.³ The burn-like purpuric vasculitis that was present in this patient has not been reported elsewhere to our knowledge.

Other Cases

The following patients who were treated by the authors have all had the classical serologic abnormalities of IM.

- A 20-year-old college student who had three major motor seizures during the course of IM.
- An 18-year-old college student who had hyperesthesia of the bridge of the nose for three weeks during an episode of IM.
- An 18-year-old college student whose hematocrit dropped to 20 as a manifestation of temporary bone marrow shut down. Fortunately, recovery ensued after she nearly died.
- An 18-year-old freshman college student who had a prolonged bout of IM that lasted two months and was manifested by a persistently high fever and

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Figure 1. Four-extremity purpura seen in Case One.

severe sore throat. She was forced to drop out of school.

- A 53-year-old male who had classical IM complicated by pneumonia and nearly died.

Diagnostic Pitfalls

Symptomatic presentations

Although 80-95 per cent of patients present with sore throat, malaise, fatigue, sweats and fever, occasional patients will have predominately gastrointestinal symptoms such as anorexia, nausea, vomiting, diarrhea, and rarely abdominal pain.^{4, 5} Infrequently, patients may present with purpura or bleeding manifestations.

Laboratory presentations

During the first week of the illness, the patient may have neutropenia and a left shift before the usual atypical lymphocytosis ensues.¹ During that time, the monospot and heterophile are negative, rarely becoming positive before the end of the first week and reaching maximum positivity during the second and third weeks. The monospot⁶ is highly accurate, but false positives and negatives may occur.^{7, 8} A diagnostic heterophile test for IM should have at least a 1:56 titer after guinea pig absorption and no titer after beef absorption.⁴ A variety of viral and bacterial illnesses can cause an

anamnestic rise in the heterophile titer for as long as one year after an attack of IM.⁹ Atypical lymphocytes can be seen in hepatitis and other viral illnesses, but the percentages are usually low.

The Epstein-Barr virus (EBV)¹⁰ has been incriminated but not proven to be etiologic for IM. The EBV has also been found in association with Burkitt's and other lymphomas, nasopharyngeal carcinoma, leukemia, and sarcoidosis. A rising titer may help confirm the diagnosis of IM. Ten per cent of patients have heterophile negative IM and are usually, but not always, EBV negative. EBV antibodies persist for years and possibly for a lifetime.

Serious Problems

Complications

Numerous serious complications associated with IM have been reported but occur quite infrequently, probably in less than 1 per cent of cases. The most common is splenic rupture, and this should be suspected if the patient has left upper quadrant pain, which is rarely a feature of uncomplicated IM.¹¹ Surgical consultation and appropriate diagnostic studies should be requested promptly because potentially fatal bleeding can occur. All patients who have an IM-like clinical picture should have gentle splenic palpation in order to avoid iatrogenic rupture. These patients are well advised to avoid trauma if possible, although splenic rupture can result even from trivial strains during the first three weeks of the illness.¹¹

A number of neurologic complications have been reported including mononeuritis, facial nerve palsies, Guillain-Barré syndrome, myelitis, meningoencephalitis, seizures, and psychiatric disorders.¹²

Other serious complications that have been reported include cardiac (myocarditis, pericarditis), respiratory (acute airway obstruction, pleural effusion, and parenchymal lung changes), and gastrointestinal (liver failure, pancreatitis). Some patients with prolonged illness may lose a job or drop out of school.

IM is rarely fatal. In one review,¹³ 87 cases were reported, although only 20 were unequivocally IM. The incidence of fatalities is probably less than one in 3,000 cases. Death most commonly results from splenic rupture or the Guillain-Barré syndrome with its concomitant respiratory insufficiency. Steroids have been recommended for the neurologic complications of IM, respiratory obstruction, hemolytic anemia, and severe thrombocytopenia, but long-term administration with its attendant complications should be avoided whenever possible. For bleeding

(Continued on page 124)



The Inconstant Certainties

Observers of medical behavior have been working overtime in recent years to accommodate the principles of morals and ethics to the shifting medical patterns — or vice versa. Sometimes the problems have been of a practical nature and consequently amenable to pragmatic interpretations and productive of only transient pain. Increasingly, however, intellectual pressures have been producing what we look on as more complicated moral issues than have ever occurred before, and a polarization has developed between those who would force the moral concepts into a form compatible with the issue and those who would deny or abolish the effort since they can accept no reconciliation of the emerging conditions with their own moral and ethical dictates. At the least, these extremes demonstrate that we have failed to achieve the ideal of absolute morality and ethos which should be inviolate without need for revision after each day's work is done.

Efforts from either direction to guide us in the ways of righteousness by firm interpretations of the eternal verities have been further complicated by the fact that current communicative methods not only disseminate changes rapidly, but bring into the conflict many individuals whose relationship, either medical or philosophic, is at best peripheral, thereby producing a body of instant experts. The result is a continuing atmosphere of contention between charges of spiritual corruption and stultifying restriction to intellect, a salubrious climate for governmental and other regulatory groups to impose their interpretations (abetted, in the case of the former at least, by the fact that they are often paying the bill). Nor is the matter made easier by the fact that before we have assimilated one element of progress, we have moved on to a new one.

If there are, currently, misgivings regarding the eventualities of our technologic successes (outstripping, as they are, the social adaptation to them), it should be remembered that no major medical advances (to our imperfect memory, at least) have occurred without exciting resistance from some

quarter as being contrary to the will of whichever almighty the objectors supported. Even the advent of the sulfas in the 30s and penicillin a little later was resisted by some since their success in the treatment of venereal disease invited increased exposure to sins of the flesh. The necessity to reorganize those principles of morality and ethics thought to be above reorganization has promoted a sense of threat and disquiet — until the resulting benefits neutralized them or the effort was abolished by subsequent failures or introduction of yet newer methods.

The comforting limitations that once existed for the medical profession — and society — made it possible to retain some sense of stability about the established moral principles, a sense of noble futility perhaps, still cited by the not infrequent reproductions of Fildes' "The Doctor." One chanted the incantations or applied the available therapies and beyond that, the physician — and society — were relieved of responsibility — success brought its comfort, or failure confirmed a higher will.

If there is one point that at once separates the medical profession of today from its predecessors and promotes anxiety in the face of current successes, it is the loss of that comfort. We know we can bring life or extend life in ways that were purest fantasy not long ago. We recognize that there are points beyond which further effort is futile, but they are distressing because we know that before long we might have the capability to displace them still farther into the future — when, of course, we will confront new limitations. But the comfort one might feel from present accomplishment and acceptance is no longer a medical matter alone, but a social and economic — even political — matter. A clear manifestation of the change in this concept of medical capacity is that we can no longer withdraw as we might have a few generations ago but must add the interpretations of these elements of society to the medical, not only in making the decision (that may still be primarily medical, although spoken with caution) but also in carrying it out.

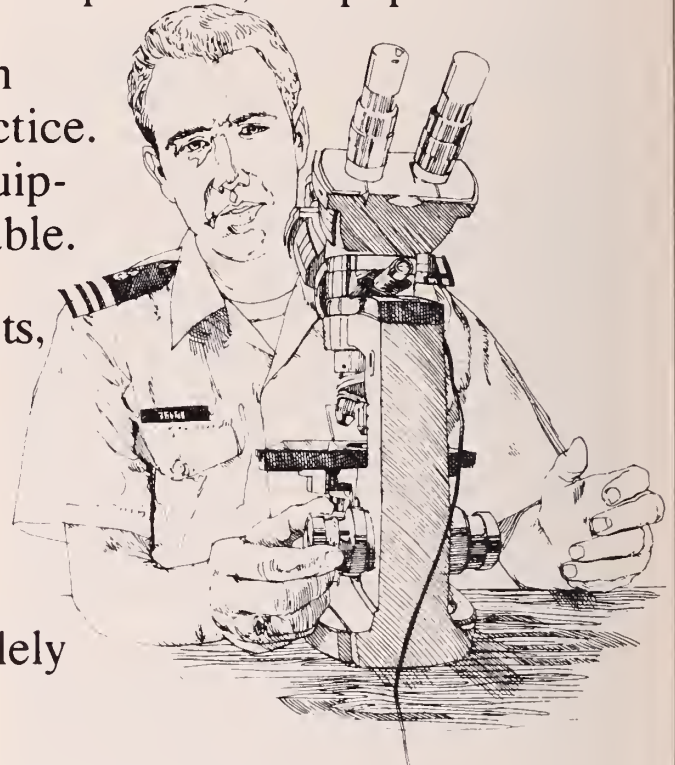
So the effort to bring morality to medicine in some absolute form continues to be the matricial binder of the medical purpose — at once the stimulus and the restraint. And our progeny, struggling with future questions, will look back in envy at the simplicity of our times. — D.E.G.

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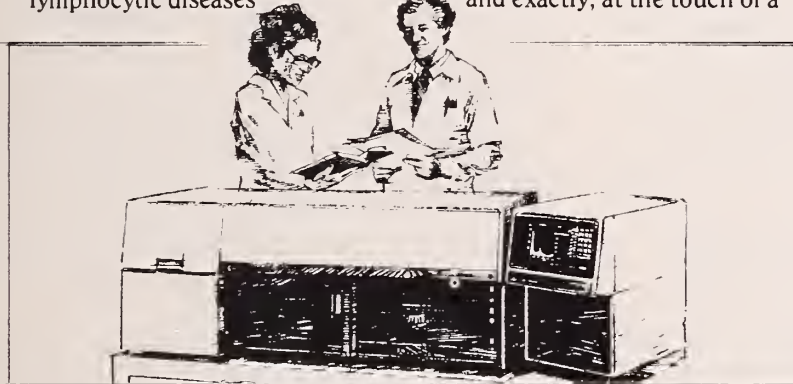
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Anesthesia

(Continued from page 109)

field,⁶ this has not been a problem at this institution. Moreover, the shunt acts as an excellent stent while closing the vessel, especially when it is small. Clotting within the shunt, embolization, air embolus, dissection of the distal intimal flap, and plaque dislodgment persist, however, as disadvantages of this approach.

Since morbidity and mortality rates as disclosed in this study are similar for both types of anesthesia (with appropriate controls), it remains for the surgeon to assess individual patient needs, apply meticulous surgical technique, and provide careful intraoperative protection for the patient as noted. In our opinion, lacking more reliable methods of assessing adequacy of cerebral circulation during general anesthesia, the patient is best served by routine placement of shunts during the procedure.

References are available from Dr. O'Holleran, Wesley Medical Center, 550 No. Hillside, Wichita KS 67214.

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Hormonal Patterns

(Continued from page 112)

oligomenorrhea is associated with low body weight, the oral progesterone withdrawal test is the initial study.

Knowledge of the hormonal profile can aid in appropriate management. For example, those with the PO pattern may be given a periodic withdrawal flow with intermittent progesterone (Provera, 10 mg/day or Norlutin, 5 mg/day orally for five to seven days). If hirsutism is a significant problem, and if the woman has contraceptive needs, the oral contraceptive pill is a good choice to suppress ovarian androgen synthesis and to elevate sex hormone binding globulin and decrease "free" or unbound circulating androgens. If the DHEA-S is elevated, a nighttime dose of dexamethasone (0.37-1.0 mgm) or prednisone (5-10 mgm) can be used. The oral contraceptive pill, of course, may also suppress DHEA-S somewhat, and better adrenal-ovarian suppression can be obtained by the combination of the oral contraceptive pill and nighttime corticosteroids. Clomiphene (Clomid) is useful in the induction of ovulation in the patient with polycystic ovary syndrome; 25 mg/day for five days is a common initial dose schedule. Women with adequate estrogen levels and a positive oral progesterone withdrawal test are most likely to respond to Clomid.¹⁴ Spironolactone (Aldactone), 25-200 mgm/day, is anti-androgenic, probably active at the hair follicle, decreasing further hair growth and acne formation.¹⁵ If a progestin or spironolactone is prescribed on a regular basis, the patient must be advised of possible risks of teratogenicity if pregnancy results concomitant to use of these medications. A diaphragm or another mechanical method is advised if contraception is needed.

Women with elevated PRL levels are asked to discontinue the oral contraceptive pill if they are using it, and the PRL is rechecked in six weeks. Those with greater elevations of PRL and a reversed LH/FSH ratio are initially further evaluated with a lateral skull film for sellar size to rule out a macroadenoma. Further investigation requires polycystoidal tomography or a CAT scan with contrast media enhancement. TRF testing may also be helpful as women with prolactinomas often have a blunted PRL response to TRF.¹⁶

Bromocryptine (Parlodel) has been remarkably effective in lowering PRL and restoring fertility in hyperprolactin women. The usual dose is 2.5 mg twice daily. It is approved by the Food and Drug Administration for use in women without evidence

of pituitary adenomata, although several case reports describe dramatic shrinkage of even large prolactinomas on this medication.¹⁷

Women students with only a mild elevation of PRL are asked to discontinue the oral contraceptive pill and other drugs known to influence PRL regulation. Weight loss is encouraged. Very few of the women with the HPRL-PO pattern here have been interested in immediate fertility. Several authors have suggested the use of nighttime corticosteroid in these patients; others have used Parlodel.¹⁸ Intermittent medroxyprogesterone (Provera) has been used here to prevent endometrial hyperplasia.

Women with HP-Dys should also avoid use of the oral contraceptive pill, as hypothalamic suppression is a potential problem, especially if fertility is desired. Restoration of more normal body weight, resolution of stress, and reduction of exercise drain, may result in more spontaneous ovulation. Women with anorexia nervosa also fit in this category. Human menopausal gonadotropin (Pergonal) may be useful in this group if fertility is desired; more recently, repetitive doses of LH-RF have been utilized to induce ovulation.¹⁹

References are available from Dr. Reith, Watkins Memorial Hospital, Student Health Service, The University of Kansas, Lawrence KS 66045.

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Mono

(Continued from page 116)

resulting from thrombocytopenia, packed red cells and rarely platelets may be indicated in dire situations. Unfortunately, platelet transfusions frequently stimulate antibodies thus limiting their utility.¹⁴

Serious Diseases That Mimic IM

A serious hematologic disorder that mimics IM is acute lymphocytic leukemia (ALL). Patients may present with leukocytosis, thrombocytopenia, anemia, and an immature appearing bone marrow. Treatment of such patients with chemotherapeutic agents could produce disastrous results. Patients who have IM do not have an increased incidence of ALL, lymphoma or other malignancies, but cases of concurrent IM and ALL have been reported.¹⁵ In one series, long-term survivors had higher anti-EBV titers than did a control group, but this has not been confirmed, nor is there evidence that IM protects against ALL.¹⁶ The diagnosis of ALL should never be made without the presence of blast cells.

Because lymphadenopathy is a major feature of IM, lymph node biopsy may be performed to rule out lymphoma. Although the reticular lymphoblast seen in IM nodes may resemble the Reed-Sternberg cell which is diagnostic of Hodgkin's disease,¹⁷ other nodal pathologic criteria and the serologic diagnostic

studies for IM enable the clinician to make the distinction between the two diseases. Posterior cervical adenopathy is more characteristic of IM than lymphoma. IM has been rarely reported to be complicated by aplasia.¹⁸

IM can be confused with viral hepatitis and chronic active hepatitis. Enzymes may normalize more quickly in IM than viral hepatitis.⁷ The specific tests for IM should be obtained prior to the consideration of a liver biopsy or treatment with steroids.

Throat cultures should be ordered for all patients with IM because of possible coexistence with strep.¹⁹ The risk of rheumatic fever, glomerulonephritis and rarely sepsis and death²⁰ exists if streptococcal pharyngitis (SP) is not adequately treated. Mere examination of the throat will not allow an accurate diagnosis. IM also suppresses the typical leukocytosis and neutrophilia of SP⁷ making the diagnosis even more difficult. Patients with both SP and IM should be treated with an antibiotic other than ampicillin, because of the high incidence of rash that develops in IM patients treated with that drug.

The clinician should be aware of the differential diagnosis in order to rule out serious diseases that mimic IM so that inappropriate and potentially dangerous therapy can be avoided.

References are available from Dr. Rubinowitz, Denver Clinic, 701 E. Colfax Ave., Denver CO 80203.

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**BP-SPEC
1983**

An added complication... in the treatment of bacterial bronchitis*

Increasing incidence
of ampicillin resistance in
Haemophilus influenzae

Ampicillin Resistant
Haemophilus influenzae

H. influenzae

S. pneumoniae

Brief Summary. Consult the package literature for prescribing information.

Indications and Usage: Cefaclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae* (*Diplococcus pneumoniae*), *Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci). Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Cefaclor.

Contraindication: Cefaclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Cefaclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out.

Precautions: General Precautions—If an allergic reaction to Cefaclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of Cefaclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when anti-globulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Cefaclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Cefaclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinistest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in terrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Cefaclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers—Small amounts of Cefaclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one

Some ampicillin-resistant strains of *Haemophilus influenzae*—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Cefaclor.¹⁻⁶

In clinical trials, patients with bacterial bronchitis due to susceptible strains of *Streptococcus pneumoniae*, *H. influenzae*, *S. pyogenes* (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Cefaclor.⁷

Cefaclor®

cefaclor

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hour. The effect on nursing infants is not known. Caution should be exercised when Cefaclor® (cefaclor, Lilly) is administered to a nursing woman.

Usage in Children—Safety and effectiveness of this product for use in infants less than one month of age have not been established.

Adverse Reactions: Adverse effects considered related to therapy with Cefaclor are uncommon and are listed below.

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100), Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis, arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Cefaclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transient abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information to the physician.

Hepatic—Slight elevations of SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

[061782R]

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.

Note: Cefaclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, light-headedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, e.g., excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. **Adults:** 30 mg usual dosage; 15 mg may suffice in some patients. **Elderly or debilitated patients:** 15 mg recommended initially until response is determined.

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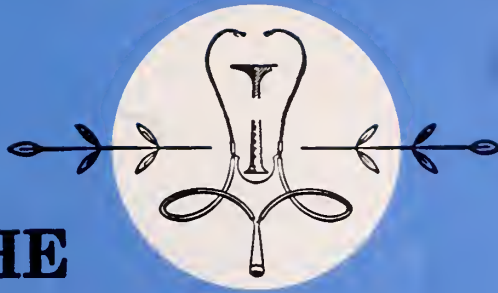
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The President's Message

As your new President, I am confronted with the formidable task of following Dr. Jimmie Gleason, who has done an outstanding job as KMS President. We enjoyed an excellent, well-attended Annual Meeting in Hutchinson, with much discussion centering on today's challenges to medicine. The resolutions passed by the House of Delegates will appear in the July issue of *The Journal*.

I wish to recognize the fine work done by Steve Carter these past two years. We are sad to lose him, but he has chosen a career of teaching, and will earn his doctorate in political science at the University of Kansas. Our longtime friend and advocate, Jerry Slaughter, will return as the fulltime Executive Director.

I plan to utilize this page to keep you informed on the activities of your state medical organization. Much is happening, and it is happening quickly. It is imperative that we, as physicians, keep in touch with each other, with our patients, and with the public. If you have a particular concern that you want to share, a question to ask or a suggestion to make, please call the Medical Society on the incoming WATS line: 1-800-332-0156.

Finally, I want to thank you for your help in the coming year. Medicine is a unique profession composed of extraordinary individuals. I am continually amazed at how much time and effort, wisdom and experience are donated freely in voluntary service by the physicians all across our state.



Medicine is unique in another way. Even as we grapple with the challenges of meeting virtually incessant and yet indefinite public demands with limited resources, our ultimate goal is clear. Each day, we physicians individually are faced with the question, "What is the best for the patient?"

We in the Kansas Medical Society must be guided by the question, "What is best for the health of the people in Kansas?"

F. Calvin Buzby, M.D.
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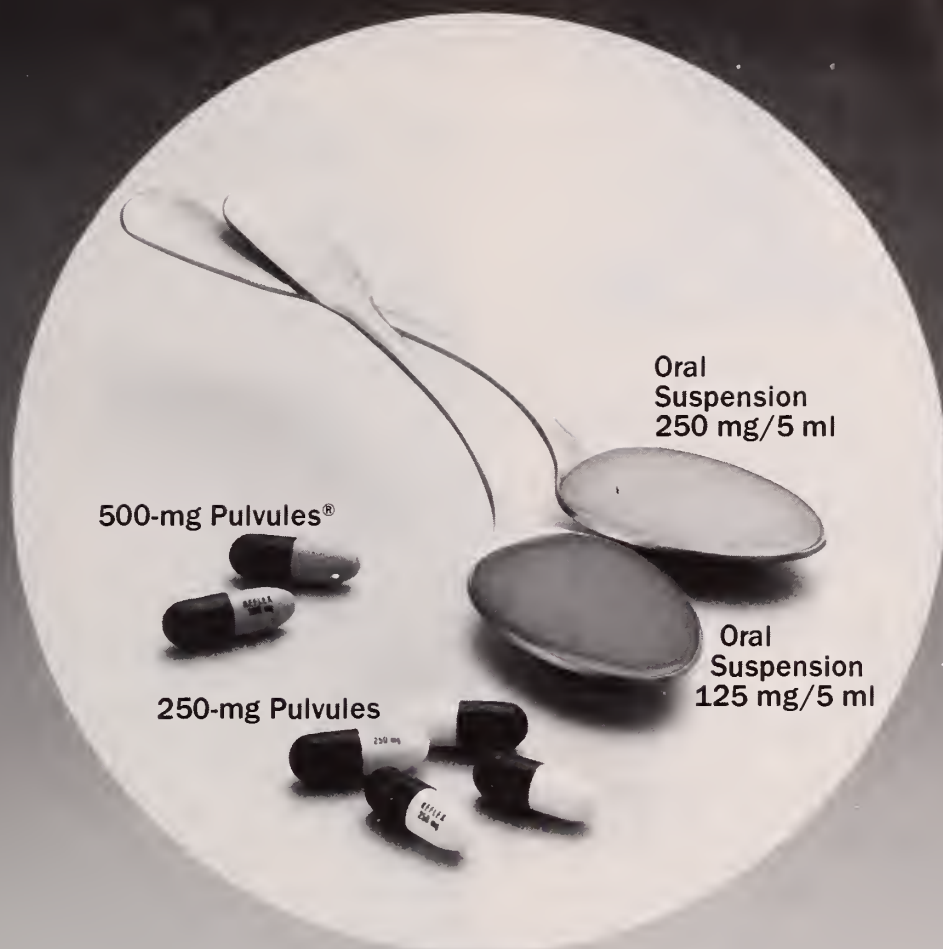
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References:

1. Stone PH, Turin ZG, Muller JE. Efficacy of nifedipine therapy for refractory angina pectoris. *Am Heart J* 104 672-681, September 1982.
2. Antman E, Muller J, Goldberg S, et al. Nifedipine therapy for coronary artery spasm. Experience in 127 patients. *N Engl J Med* 302 1269-1273, June 5, 1980.

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2. **Chronic Stable Angina (Classical Effort-Associated Angina):** PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: Excessive Hypotension: Although in most patients the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers, if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients usually receiving a beta blocker have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: General: Hypotension: Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug interactions: Beta-adrenergic blocking agents. (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long-acting nitrates. PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

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ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients; transient hypotension in about 5%, palpitation in about 2%, and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in less than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LOH, SGOT and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

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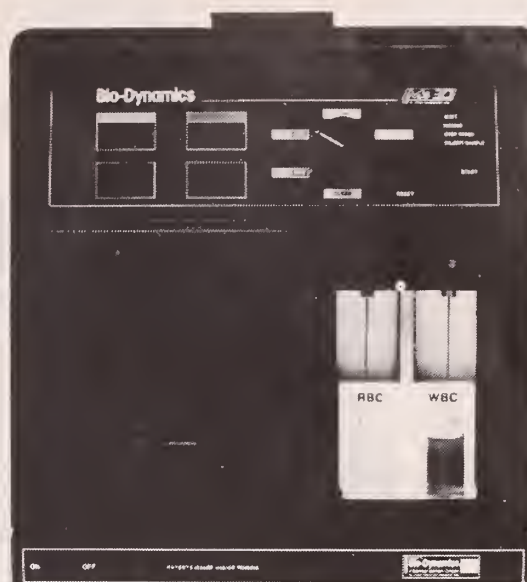
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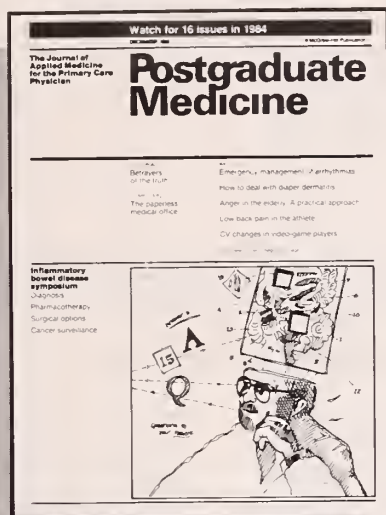
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Exocrine Pancreatic Cancer at UKSM

DAN A. WAXMAN, M.D. and PATRICK J. FITZGERALD, M.D., *Kansas City, Kansas*

PANCREATIC CANCER now causes the death of more than 22,000 persons each year in the United States. It is the fourth leading cause of cancer deaths in men and the sixth most common cause of cancer deaths in women,¹ and it appears to be increasing in incidence throughout much of the Western World. Relatively little is known about its etiology except that excessive cigarette smoking increases the risk, most cases occur in the elderly, and diabetics appear to be at increased risk for this cancer. Therapy is relatively ineffective, and less than 1 per cent of patients survive for five years after diagnosis.²

Recently a detailed morphological classification of cancer of the exocrine pancreas has been proposed with the hope that a subtype(s) might be related to a distinctive etiological factor, may have a better prognosis, or might respond to a particular therapeutic regimen (*Table I*). Since these cases were collected from a large northeastern U.S. urban cancer hospital, Memorial-Sloan Kettering Cancer Center (MSKCC), the question has been raised as to how well the classification reflects the incidence of various types of pancreatic cancer in the overall population. The cases at UKSM-KC might better reflect the incidence of types of pancreatic cancer found throughout the country.

We have examined the records of all cases of pancreatic cancer that came to autopsy at the Department of Pathology, University of Kansas School of Medicine (UKSM) to determine the types of pancreatic cancer and the relative frequency of these

types in a university teaching hospital in a central part of the United States. The results were then compared to the types and their relative frequency at the MSKCC.

Methods

The autopsy report of each case of cancer of the pancreas listed in the autopsy files of the Department of Pathology from 1952 through 1982 inclusive was examined and all tissue sections of each case were inspected. The files of the Tumor Registry, UKSM, were examined for the same years and used as a check list for cases in the autopsy files. The names of all patients with a diagnosis of cancer at UKSM since 1947 have been recorded in a Central Tumor Registry.

Surgical pathology reports, tissue sections of biopsies and resections of the pancreas, and hospital records of patients who came to autopsy were examined. Cases of rare cancers of the pancreas were sought from Surgical Pathology, Department of Pathology, UKSM. Formalin fixation, paraffin embedment, and hematoxylin and eosin staining of tissues were used for routine processing of tissue. Special stains and the recutting of blocks of tissue were performed when necessary.

Results

There were 98 cases of ampullary and pancreatic cancer in the 11,560 autopsies performed at UKSM Department of Pathology from 1952 to 1982. The number of patients is given under each type of a cancer in the three main categories: Pancreatic ductal carcinoma; ampullary-biliary duct cancer; and islet-ductular carcinoma (*Table II*).

From the Departments of Pathology and Oncology, The University of Kansas School of Medicine-Kansas City.

Address reprint requests to Dr. Waxman, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.

TABLE I
PRIMARY MALIGNANT NEOPLASMS OF THE NONENDOCRINE PANCREAS*

	<i>Patients Number (%)</i>		<i>Patients Number (%)</i>
DUCT (DUCTILE) CELL ORIGIN	573 (88.8)	CONNECTIVE TISSUE ORIGIN	4 (0.6)
Duct cell carcinoma (494)		Leiomyosarcoma (1)	
Giant cell carcinoma (27)		Malignant fibrous histiocytoma (1)	
Giant cell carcinoma (osteoclastoid type) (1)		Malignant hemangiopericytoma (1)	
Adenosquamous carcinoma (20)		"Osteogenic" sarcoma (1)	
Adenosquamous (spindle cell) carcinoma		Fibrosarcoma	
Microadenocarcinoma		Rhabdomyosarcoma	
(solid microglandular) (16)		Malignant neurilemoma	
Mucinous ["colloid"] carcinoma (9)		Liposarcoma	
Cystadenocarcinoma (mucinous) (5)		UNCERTAIN HISTOGENESIS	59 (9.2)
Papillary cystic tumor (1)		Pancreaticoblastoma (simple type)	
Mucinous-carcinoid carcinoma		Pancreaticoblastoma (mixed type) (1)	
Carcinoid		Unclassified (58)	
Oncocytic carcinoid		Large cell (50)	
"Oat-cell" carcinoma		Small cell (7)	
Ciliated cell carcinoma (?)		Clear cell (1)	
ACINAR CELL ORIGIN	8 (1.2)	MALIGNANT LYMPHOMA (?)	
Acinar cell carcinoma (7)		Histiocytic	
Acinar cell cystadenocarcinoma (1)		Plasmacytoma	
MIXED CELL TYPE	1 (0.2)	Total	645
Duct-islet cell (1)			
Duct-islet-acinar cell			
Acinar-islet cell			
Carcinoid-islet cell			

* Classification of malignant lesions of the pancreas and the relative frequency of the types at Memorial Hospital, New York, NY. Figures obtained from over 500,000 surgical specimens and 13,882 autopsies. 821 patients were listed as having pancreas (non-islet) cancer; adequate clinical and pathologic material was available for study in 645 patients. Diagnoses without (numbers) indicate that such a cancer did not occur in the Memorial Hospital patients during the years of the review (1949-1978), but has been reported in the literature or was seen by us subsequent to 1978.

Demographic Data

Age, Sex, and Color

PANCREATIC DUCTAL CANCER: The 77 patients with pancreatic ductal adenocarcinoma were 47 men and 30 women, comprised of 38 white men, nine black men, 21 white women, and nine black women. The median age of all patients was 66 years, that of all men 66.5 years and of all women 66 years. The black patients had a median age of 66.5 years. Black men had a median age of 68.5 years, and white men 66.5 years. The median age of white women was 69.5 years, and that of black women 66 years. The range of ages overall was from 34 to 93 years.

The male to female ratio of all patients was 1.6, but in white patients it was 1.7 and in black patients it was 1.0.

Black patients made up 23 per cent of pancreas cancer patients; black men comprised 19 per cent of

TABLE II
UKSM AUTOPSY STUDY
1952-1982 11,560 autopsies

NEOPLASMS OF THE PANCREAS		
I. PANCREATIC DUCTAL ORIGIN		87
Ductal cell adenocarcinoma	77	
Giant cell carcinoma	6	
Adenosquamous carcinoma	3	
Unclassified	1	
II. AMPULLA-BILIARY DUCT ORIGIN		3
Ampulla of Vater	1	
Common bile duct (pancreas region)	2	
III. ISLET-DUCTULAR CELL ORIGIN		8
Islet cell carcinoma	6	
Carcinoid-islet carcinoma	2	
Total		98
IV. BENIGN NEOPLASMS		
Islet cell adenoma	12	
Serous cystadenoma	2	
Mucinous cystadenoma	1	
Total		15

all male patients and black women comprised 30 per cent of female pancreas cancer patients.

GIANT CELL CANCER: Of the six cases, four were men and two were women. Two men were white, one was black, and their median age was 62 years. One male patient was a Mexican-American of 47 years. The women were white, 52 and 59 years of age.

ADENOSQUAMOUS CARCINOMA: There were three patients, two men and one woman, and all were white. The men were 63 and 70 years of age and the woman 52 years.

ISLET CELL CANCER: There were six patients with islet cell cancer, all were white, five were men and one was a woman. The median age for the men was 56 years and the woman was 78 years of age. Five patients had metastases, and the sixth — the female patient — had a small nodule in the head of the pancreas, 0.8 cm in diameter. In the latter case there was perineural invasion as well as intrapancreatic spread.

Pathologic Data

Site, Size and Stage

The head of the pancreas was the principal site of the cancer in 65 per cent of patients, the body in 8 per cent, the tail in 3 per cent, the body and tail in 13 per cent, and some combination of head, body and tail in 11 per cent.

The size of the neoplasms varied from very large tumors 10-15 cm in diameter seen with the giant cell carcinoma to a 1.5 cm lesion found incidentally in a patient who was stabbed to death. Staging was not present in all protocols and often only a few lymph nodes were examined, but in all but four patients there were multiple nodes involved and/or distant metastases present. One of the latter four died post-operatively after a Whipple resection with no evidence of residual cancer, and in the other three patients the cancer invaded locally the ampulla, duodenum, or the common bile duct.

Types and Relative Frequency

Table II shows the classification, the number of patients, and the relative frequency of the types of pancreatic cancers found at UKSM.

It is apparent that ductal cell adenocarcinoma made up a high percentage of cases (79%).

Patients with giant cell carcinoma occurred in 6 per cent of pancreatic cancers. Adenosquamous cell carcinoma occurred in 3 per cent of pancreatic cancers. Acinar cell carcinoma and sarcoma were not

present in the cases studied. One case was a combination of papillary carcinoma, mucinous adenocarcinoma, and areas of solid carcinoma and was put in an unclassified group.

Islet cell cancers and cancers of the ampulla and common bile duct in the region of the pancreas made up 11 per cent of the total cancer cases.

Benign neoplasms made up 13 per cent of all neoplasms of the pancreas, most being islet cell adenomas. The cystoadenomas were rare.

Discussion

It is well recognized that autopsy findings over a long period of time do not necessarily correctly reflect the incidence of a disease, particularly if a disease has a significant rate of cure, if the incidence of disease changes, or if the disease permits the patient to live for many years and die of another disease. The majority of patients with pancreatic cancer, however, die within one year of the diagnosis of the cancer, and only a small percentage of patients live for two years, so that these factors are probably not so significant in this disease.

When a very high percentage of the cancers of an organ is represented by a major type, a large number of cases is needed to cover the gamut of the rare and very rare types. A MKSCC study of 2587 autopsies during a six-year period (*Table III*) did not reveal many of the rare cancers present in the combined grouping of autopsy and surgical pathology cases for a long span of time (*Table I*). Even the relatively large number of cases from a cancer hospital did not reflect all types of cases reported in the literature as shown in *Table I*.

The mean age of patients with pancreatic cancer at UKSM was within the range of most reports.³ The black male to black female ratio of 1.0 is lower than the overall 1.5 reported in most studies. The ratio of white men to white women (1.7) is in the range generally reported. There are studies that report that black women have a higher incidence of pancreatic cancer than white patients.⁴

In a comparison of the frequency of the types of non-islet pancreatic cancer between UKSM and MSKCC (*Table IV*), the duct (ductule) cell adenocarcinoma and the giant cell carcinoma made up most of the cases — 96 per cent of patients in each study. At UKSM, acinar cell carcinoma, papillary-cystic tumor, microadenocarcinoma, and sarcoma were notably absent.

A wider selection of morphological types is generally present in surgical pathology than in autopsy specimens, and amongst the rare types of exocrine pancreatic neoplasms present in the Surgi-

TABLE III
AUTOPSY STUDY OF PANCREAS*
2587 Autopsies — Pancreas Involved in 411 Patients (16%)

<i>Involvement of Pancreas at Autopsy</i>	<i>Patients Number (%)</i>	<i>Involvement of Pancreas at Autopsy</i>	<i>Patients Number (%)</i>
BENIGN "TUMORS"	4 (0.1)	SYSTEMIC MALIGNANT NEOPLASMS INVOLVING THE PANCREAS	67 (2.6)
Cyst (1)		Malignant lymphoma	41 (1.6)
Cystic pancreas (1)		Hodgkin's disease	7 (.03)
Islet cell adenoma (1)		Leukemia	19 (1.0)
Serous Cystadenoma (1)		METASTASIS TO PANCREAS FROM CANCER OF:	261 (10.1)
PRIMARY MALIGNANT NEOPLASMS	63 (2.4)	Breast (51)	
Duct cell carcinoma (51)		Lung (49)	
Giant cell carcinoma (6)		Malignant melanoma (skin) (23)	
Islet cell carcinoma (4)		Stomach (19)	
Acinar cell carcinoma (1)		Colon (19)	
"Osteogenic" sarcoma (1)		Ovary (13)	
INVASION OF PANCREAS BY CANCER OF ADJACENT ORGAN	16 (0.6)	Uterine cervix (12)	
Stomach (6)		Esophagus (10)	
Colon (3)		Neuroblastoma (various sites) (9)	
Duodenum (2)		Other sites (56)	
Ovary (2)		Total	411 (16)
Gastroesophageal junction (1)			
Gallbladder (1)			
Retroperitoneal neurofibrosarcoma (1)			

* From the files of Memorial Hospital, New York, NY, 1973-1978.

cal Pathology Division were: Papillary-cystic tumor, mucinous cystadenocarcinoma, and oncocytic carcinoma. The last case is unique and indicative of the variability with small numbers for it was not found in the larger MSKCC group (*Table I*).

Some rarities not present in the MSKCC cases were seen in the UKSM autopsies. In a predominantly giant cell carcinoma of the pancreas there was a large fraction of the tumor tissue that had a significant adenosquamous carcinoma component. It has been shown² that small foci of morphologic types (clones?) of pancreatic cancer exist in most cancers of this organ, that usually one type predominates in the tumor when it is recognized clinically, and that it is this opportunistic type that ordinarily gives rise to invasion and metastases. However, two morphologic types may be equally predominant both in the primary and metastatic cancers.

In a 72-year-old male patient, two different carcinomas were apparent: One was a squamous cell carcinoma of the lung with metastasis to the hilar nodes, right adrenal, cerebral hemispheres, cerebellum, basal nuclei and pons; the other was a mucinous adenocarcinoma of the head of the pancreas which infiltrated and obstructed the biliary and pancreatic ducts, extended into the peripancreatic lymph nodes, and gave rise to metastases in the liver. There was no

overlap of morphologic patterns as these two metachronous cancers went their separate classical pathways.

Although statistically there is a close relationship between the size of the pancreatic cancer and the relative prognosis,⁵ the findings at autopsy of a non-obstructing cancer of the pancreas 1.5 cm in diameter, in a 51-year-old man stabbed to death, and

TABLE IV
COMPARISON OF TYPES OF PANCREATIC
CANCER (NON-ISLET) AT AUTOPSY

<i>Type</i>	<i>UKSM*</i>		<i>MSKCC†</i>	
	<i>#</i>	<i>%</i>	<i>#</i>	<i>%</i>
Ductal adenocarcinoma	77	89	51	86
Giant cell carcinoma	6	7	6	10
Adenosquamous carcinoma	3	3	0	—
Acinar cell carcinoma	0	—	1	2
Sarcoma	0	—	1	2
Unclassified	1	1	0	—
	87	100	59	100

* UKSM = University of Kansas School of Medicine, Department of Pathology, 11,650 autopsies (1952-1982 inclusive).

† Memorial Sloan-Kettering Cancer Center, 2,587 autopsies (1973-1978).

the presence of metastases to lymph nodes in the porta hepatis are very instructive. In contrast, there was one patient in whom no invasion of lymph nodes or metastases was found; the patient died in diabetic coma and had a 4 cm nodule of tumor associated with invasion but not obstruction of the pancreatic and common bile ducts.

In two patients who had a Whipple resection there was carcinoma in situ in the residual pancreas; there has been a report that the incidence of carcinoma in situ is as high as 38 per cent.⁶

Two patients with pancreatic ductal cancer had an associated genetic disorder. One was a 47-year-old Mexican-American who had a carcinoma of the head of the pancreas, partly giant cell carcinoma and partly adenocarcinoma, who also had epidermolysis bullosa dystrophica with corneal opacities, depigmentation of the skin of upper and lower extremities, and partial absence of fingernails. This lesion is a genodermatosis in which squamous cancer of the skin has sometimes been associated with the secondary scarring of digits of the hands and feet.⁷ The presence of pancreatic cancer at a relatively young age emphasizes the genetic aspect. The second patient was a white man 34 years of age with a history of familial pancreatitis (three uncles with pancreatitis). The patient also had chronic pancreatitis with pancreaticolithiasis. He also was much younger than the mean age for pancreatic cancer. We found no reference in the literature to an association of epidermolysis bullosa dystrophica with pancreatic cancer, but an increased incidence of pancreatic cancer is reported in patients with familial pancreatitis.⁸

The capriciousness of small numbers is illustrated by the presence in the UKSM surgical pathology files of a cancer of oncocytic carcinoma, reported by Huntrakoon⁹ which, to date, appears to be unique.

Ampullary and Biliary Duct Cancer

As in most studies¹⁰ the ampullary and biliary tumors were found to make up a small percentage of the ampulla-head-of-pancreas region. A biliary duct cancer of the common bile duct in the region of the head of the pancreas occurred in one of the youngest patients in the study, a 36-year-old American Indian woman. The histology was that of a biliary duct adenocarcinoma. This group of patients is known to have a high incidence of gallstones and gallbladder cancer; but neither was present in this patient.

The ampullary cancer was of the mixed type,¹⁰ Stage III, involving the ampulla of Vater, duodenum, pancreas, and peripancreatic lymph nodes. In some types of ampullary cancer there is a better prognosis for a five year survival than for patients

with pancreatic ductal carcinoma,^{5, 10} but in none of the cases was surgical resection possible at clinical presentation.

Carcinoid-Islet Cell Carcinoma

This is a debatable group of neoplasms which morphologically resemble both carcinoid and islet cell tumors and each has neurosecretory granules.² Both patients had large areas of the tumor resembling carcinoid tumor and others suggesting islet cell tumor. Neither patient showed clinical or symptomatic signs of islet cell hyperfunction. One had an associated carcinoid tumor of the appendix as well as one of the pancreas and metastases to the liver. He had vomiting and diarrhea but no flushing, and a urinary examination for 5-hydroxy-indole-acetic acid was negative. The other had no clinical signs or symptoms of islet cell or carcinoid cell hyperfunction. No immunological examinations were performed.

Unclassified Cancer

In most series of cancers of the pancreas there is a small number, 5-10 per cent, of the total cases that cannot be classified.² In the one UKSM patient there were foci of papillary carcinoma, mucinous adenocarcinoma, solid carcinoma, and some other cell types in the neoplasms. It did not fit into any of the above categories and was listed as unclassified.

Benign Neoplasms

There was a higher percentage of islet cell adenomas in the total number of benign neoplasms at UKSM than at MSKCC. The interest, expertise and discovery of families with endocrine neoplasms by the staff^{11, 12} may be important factors in the relatively large number of cases.

Conclusion

The percentage of autopsies with exocrine pancreatic cancer was lower (0.8%) here than at MSKCC (1.7 to 2.3% during two different time periods).

There appears to be a similar distribution of the commonest types of cancer of the exocrine pancreas — ductal adenocarcinoma and giant cell carcinoma — which made up 96 per cent of all cancers of the exocrine pancreas at both institutions.

Many rare and very rare types were not present in the autopsy cases of either hospital. Some of these were noted in the respective Surgical Pathology files. In 4 per cent of the cases of pancreatic cancer there was no spread to lymph nodes and no metas-

(Continued on page 152)



Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Advice to Travelers

DANIEL R. HINTHORN, M.D.,* *Kansas City, Kansas*

INTERNATIONAL TRAVEL has become common for Americans. Travelers now are routinely consulting their personal physician for advice on immunizations needed to prepare for travel, and for prescriptions for drugs to take while traveling to prevent diarrhea and other illnesses.

This article reviews a number of suggestions that physicians can give to patients to help make travel safer and more enjoyable.

Preparation for Travel

Preplanning. Persons with one of the following medical conditions should consult a physician one month before a trip: angina, previous heart attack, emphysema, asthma, diabetes, seizures, colitis, allergies, broken filling, poorly fitting dentures, or any unexplained disorder.

Medical Information Sheet. Travelers with chronic disorders would be wise to carry a medical information sheet with them listing name, address, social security number, insurance company name and policy number, employer information, name and phone number of person to notify in an emergency, blood type, brief report from the physician outlining medical disorders and abnormal test results, current medications (include generic names), dosages, drug allergies, reasons for hospitalizations, immunizations with the dates received, and telephone number of the physician.

Traveler's Medical Kit. Suggested items will vary depending on the duration, destination of the trip, and previous illnesses of the traveler (*Table I*). The

following items should be considered: extra pair of eye glasses, band aids, ace wrap, antiseptic (Betadine), scissors, tweezer, teaspoon, tablespoon, thermometer, flat toilet paper, soap, wash cloth, wash and dry towelettes, aspirin (or acetaminophen), sunscreen containing PABA, motion sickness pills such as Dramamine, antihistamines, cotrimoxazole and Pepto-Bismol for therapy of traveler's diarrhea, sleeping pills for dealing with changes in time zones, water purification tablets (see discussion of prophylaxis below), salt tablets if travel is to include the tropics, sugar sweeteners if desired, insect repellent (towelettes impregnated may be easiest to transport), mosquito netting for those who will spend time in rural malarious areas.

Patients with chronic illness should take sufficient digitalis, insulin, anticoagulants, dilantin, or other medications. Drugs sold abroad may vary in name, potency, or bioavailability. Travelers should be warned that over-the-counter drugs may not carry

TABLE I
ITEMS TO INCLUDE IN TRAVELER'S
MEDICAL KIT

Extra eye glasses and prescription
Band aids, ace wrap
Topical antiseptic
Scissors, tweezers, thermometer
Flat toilet paper, soap, towelettes
Aspirin or acetaminophen, antihistamines
Sleeping pills
Sunscreen, motion sickness pills
Halazone tablets or iodine for water purity
Salt tablets, nonsugar sweeteners
Insect repellent, mosquito netting
Chloroquine or Fansidar
Sulfamethoxazole-trimethoprim or doxycycline
Pepto-Bismol

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From the Department of Internal Medicine, UKSM-KC.
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hazardous label warnings: chloramphenicol, sulfas, or butazolidin may be included in cold or antidiarrheal preparations.

Immunizations. Immunizations required for international travel are cholera and yellow fever for anyone who travels from or through an area with endemic disease. Diphtheria and tetanus should have been given within the previous ten years. Travelers to third world countries should have had oral trivalent polio vaccine, and should receive gamma globulin every four months to prevent hepatitis. Medical workers who may have contact with blood products should consider taking the hepatitis B vaccine.

International Certificates of Vaccination must be printed and completed in English or French. These are individual certificates carried by each traveler. The physician must sign the certificate, write out the month and the date (*e.g.* May 9, 1975), and validate the certificate using the uniform stamp. Failure to comply could mean detention in some countries, and even immunization or quarantine. Unless there is an outbreak of smallpox, yellow fever, or cholera in one of these countries, no immunizations are required when traveling directly between any of the following countries: United States, Europe, Canada, Mexico, and the Caribbean countries.

Rabies vaccine given pre-exposure is recommended for persons at high risk of exposure to wild animal populations or dogs. A definite risk is present in the Middle East, Asia, tropical Africa, and Latin America. Immunizations for plague and typhus are available. Plague is sporadically reported from Southeast Asia, Burma, South America, and Africa. Typhus occurs in the Himalayas, the Andes, and central-eastern Africa, areas seldom visited by Americans. Meningococcal meningitis epidemics occur in the Sahel region of Africa, and in South America. Bivalent Type A & C vaccine is available. The pneumococcal vaccine is available but does not have an exclusive travel use. Most would not recommend the routine use of tuberculosis prophylaxis with BCG.

Precautions While Traveling

Flying. Jet lag requires that the traveler readjust at a rate of about one day for each one to two hours of time change. Sedatives may be required at night to sleep.

Altitude. More frequent rest periods are suggested during the first few days after travel to high altitudes.

Water and ice. One must assume that piped water is contaminated with pathogens. Boiling water for ten minutes or chemically treating for 30 minutes with iodine tablets or Halazone should be the rule

before using water for drinking, making ice, or brushing teeth. Alternatively coke or tea can be used to brush teeth. Iodine tablets are superior to Halazone tablets for killing amoeba or *Giardia lamblia* cysts, but either would eradicate the enterotoxigenic *E. coli* or rotaviruses, two major pathogens transmitted by contaminated water in developing countries.

Food. Well-cooked food, served hot, is usually safe. Raw fruits should be eaten only if the peeling is intact, and can be peeled just before eating. Salads and raw vegetables should be avoided entirely since cysts of amoeba and eggs of helminths may contaminate them. Soaking and scrubbing lettuce leaves with iodine solutions will remove or kill most parasites. Cheeses may harbor *Brucella*.

Bites. Care should be exercised in contact with animals, both pets and wild animals, due to endemic rabies. Poisonous snakes unfamiliar to Americans may be encountered in remote areas. Insect bites of mosquitoes or flies may prove to be more than a nuisance, and transmit malaria, trypanosomiasis or leishmaniasis.

Disease Prevention

Diarrhea may be caused by a variety of microbes, nervous tension, changes in dietary intake, or excessive consumption of coffee or alcohol. Three important considerations in preventing microbial diarrheas include defensive eating and drinking, prophylactic medications, and early antibiotic therapy (*Table II*).

Enterovioform has been removed from availability in the United States since it has been associated with a syndrome of myelo-optic-neuropathy (SMON). Unfortunately, it is still available as prophylaxis for traveler's diarrhea in certain parts of the world.

Prophylactic medications have been shown to prevent traveler's diarrhea in certain settings. Pepto-

TABLE II
ANTIDIARRHEAL MEDICATIONS FOR TRAVEL

Medication	Reduce # Stools	Caution*
Lomotil	Yes	Yes
Imodium	Yes	Yes
Parepectolin	Yes	Yes
Donnagel PG	Yes	Possibly
Pepto-Bismol	Yes	No
Kaopectate	No	No

* If fever, blood or mucus in stools, illness may be made worse by some of these.

Bismol, 60 ml four times per day, beginning the first day of travel and continued for one day after returning is effective. The drug is bulky and inconvenient to transport for a long trip. Doxycycline (Vibramycin), 100 mg twice on the first day, followed by 100 mg/day has been shown to prevent diarrhea from enterotoxigenic *E. coli*. Similarly, one double strength tablet of sulfamethoxazole and trimethoprim (SMX-TMP) taken daily has prevented traveler's diarrhea. Unfortunately, bacteria in many parts of the world are becoming resistant to doxycycline and in some areas to SMX-TMP. Just how long these drugs will continue to be useful in prophylaxis is not known. Side effects are an important concern in prophylaxis with these drugs. If prophylaxis is given to most travelers — because of the large number of travelers each year — even a modest percentage having drug reactions will result in a significant number of adverse reactions.

Therefore, many investigators do not prescribe drugs prophylactically, but prefer to advise travelers to wait for diarrhea or abdominal symptoms and then begin early treatment. In patients with infectious diarrhea, SMX-TMP, one double strength tablet twice daily, or TMP, two tablets twice daily, has been shown to reduce abdominal pain, nausea, and the number of unformed stools much more rapidly than does placebo. Pepto-Bismol may have a favorable effect on diarrhea caused by enterotoxigenic *E. coli*, although large doses can increase the salicylate levels into the toxic range for persons already taking large doses of aspirin for arthritis.

Maintaining hydration during diarrhea is also important. Oral glucose, sodium, potassium solution similar to that used for rehydration of cholera patients is beneficial. This can be prepared by mixing eight ounces of fruit juice rich in potassium, one-half teaspoon of honey or corn syrup, and a pinch of table salt (using the thumb and the first two fingers). Packets containing the drug mixture are available in many third world pharmacies.

Malaria chemoprophylaxis may be the most important advice to give travelers. Adults who travel to malarious areas should take 500 mg (300 mg base) of chloroquine phosphate once weekly, beginning one week before entering and continuing until six weeks after leaving an endemic area. After returning from areas where *P. vivax* or *P. ovale* are endemic, primaquine phosphate, 26.3 mg (15 mg base), is prescribed daily for two weeks. Care must be taken not to cause hemolytic anemia in persons who are glucose-6-phosphate dehydrogenase deficient. Travelers to areas having chloroquine-resistant *P. falciparum* should take, in addition to chloroquine,

pyrimethamine 25 mg, with sulfadoxine 500 mg in the fixed combination called Fansidar (Roche). To find whether a particular destination is reported to have chloroquine-resistant *P. falciparum*, lists are published by the *Morbidity and Mortality Weekly Report* and by the World Health Organization. Currently these areas include central Africa, Central and South America, and Southeast Asia over to India. Malaria resistant to both chloroquine and Fansidar has been reported in the Amazon region of Brazil, and near the Thai-Kampuchean border. In such areas quinine sulfate, 325 mg bid, may be useful for prophylaxis in selected patients.

Schistosomiasis occurs in Africa, the Middle East, Puerto Rico, and St. Lucia in the Caribbean. Swimming or bathing in fresh water in these areas should be avoided due to the presence of the snail that carries the parasite. Tap water usually does not transmit schistosomiasis since the cercariae cannot live outside of the snail for longer than 1.5 days.

Selecting a Physician

Several mechanisms are available to select a physician overseas. The International Association for Medical Assistance to Travelers Directory may be obtained by writing 350 Fifth Avenue, New York, NY 10001. In the absence of the Directory, a large hospital affiliated with a medical school is more likely to have someone who speaks English, is familiar with US medications and diseases, and will be more likely to practice medicine as it is practiced in this country. Alternatively the US Embassy or Consulate can be approached for advice. Other sources for information about physicians who speak English include travel agencies, large airline or steamship companies, hotels, military bases, Peace Corps personnel, or the British Embassy.

Surgery should be avoided while overseas except for emergency conditions. One should beware of injections given by pharmacists in small drugstores, since sterile needles are not always used.

Advice on Returning

Many tropical infections may not be manifest for some time after the traveler has returned home: malaria, hepatitis A or B, schistosomiasis, or intestinal parasites. Any illness that develops during travel or within 12 months after return should be suspected as possibly being travel related, and the appropriate investigations performed.

A bibliography of useful information is available from Dr. Hinthorn, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.



Editorial COMMENT

Physicians generally have an uneasy feeling about lawyers. There are a few exceptions of circumstance, perhaps — when, for example, legal services are needed in maneuvers such as malpractice defense or countersuits. There are even occasional instances of social rapport when professional differences are, fortunately, muted. At the organizational level, the respective parent groups issue intermittent announcements that there are no real differences of attitude or purpose between them — and anyway they are trying to work them out.

Meantime (this to keep peace in our editorial family), a small but increasing number of renegades from both camps are acquiring the academic qualifications to serve either side but their intent seems not so much to reconcile their respective philosophies as to gain the inside track in confronting their opposite numbers. Physicians generally, as we said in the first place, have an uneasy feeling about lawyers.

Although the medical profession needs no particular reminder of the progressive intrusion of lawyers into its functions, we have recently become aware of a group whose emergence was probably inevitable, the National Health Lawyers Association. It is not surprising that such a group exists. A prime national characteristic is that when two or more individuals can find a common denominator, they feel compelled to organize, complete with constitution, by-laws, and an annual dinner meeting with the spouses. The fact is that this organization represents a sort of maturation, a recognition that matters of health are so extensive, so much a part of the national scene, and so productive of legal questions that the united interests of the legal profession demand this type of forum.

This is not in any way to object to such an organization or see deliberately ulterior motives, either of presence or purpose, in its existence. The generality of its title leaves room to believe that it may have interest — even positive benefit — for physicians, particularly those immediately involved in negotiations of medicine with various social and political entities — and that's a lot of territory. Our interest, rather, was attracted by the remarks of one of their members at a recent meeting regarding the progres-

The Odd Couple

sion of changing attitudes in the antitrust climate as it is affecting the medical corps. It was not so much the details (we need to save some of them for another time) as the implications for one limited phase of medical activity, hospital staff selection.

Physicians have, in the past, been accorded — and accepted as a basic right of the profession — the responsibility of passing on the professional merits of staff membership applications and, consequently, reappointments. It was generally assumed that, being familiar with the needs, acquainted with the intricacies of professional preparation and qualification, and generally supportive of a high quality of function, they knew how to do this best. Professional interests were better served not by prostitution of the process but by adherence to objective standards of quality. By and large, hospital boards accepted their recommendations and generally confined their attentions to the incoming bills, the administrator's ambitions, and the balance sheet.

No more. As hospitals conglomerate and medical staff activities become an administrative unit in a complex of services (albeit an important one), it is an increasing certainty in the legal mind that there is an abiding conflict of interest for physicians in choosing their peers or judging their functions, that what the physicians thought of as qualifications for such judgments were actually grounds for exclusion from it, and that only the hospital administration and board (with legal guidance, of course) had the combination of vision and objectivity to make such determinations. The idea has been fostered to some extent by disgruntled physicians denied appointment or reappointment, sometimes justified, sometimes not, for which judicial intervention has been sought, sometimes successfully, sometimes not.

The remarks referred to (and presented, as mentioned, in greater length and detail) are not so much revelation as confirmation of the situation which is well past the trend stage. Physicians have seen with varying clarity the development of such attitudes but have been relatively impotent to offset them, given the state of social pressures. Although this represents but one limited phase of the altered physician-hospital relationship, the process has reached a pervasive level: physicians will be increasingly pressed to accommodate their professional integrity to what are seen to be legal realities. At the very least, it is another expression of the direction in which medical influence is moving as the relentless repositioning of the physician in the medical effort is accomplished. Plus a need for more physicians to go to law school. — D.E.G.

Council Meeting

Report of Meeting Held March 18, 1984

The Council convened March 18, 1984, in Topeka. Councilors and alternates present were Drs. Carl D. Ambler, Franklin G. Bichlmeier, F. Calvin Bigler, Kenneth M. Boese, James G. Bridgens, John P. Brockhouse, K. William Bruner, Jr., Louis M. Culp, Herbert Fransen, R. J. Haskins, David A. Leitch, W. E. McAllaster, Warren E. Meyer, John R. Neuenschwander, James H. Ransom, Edwin D. Rathbun, Ralph R. Reed, Newton C. Smith, Linda D. Warren, Wallace N. Weber, Kermit G. Wedel, and Emerson D. Yoder. Also present were Past Presidents Drs. John N. Blank and Lucien R. Pyle; Specialty Society Representative Dr. Alan Sanders; and Resident, Dr. Catherine Willner. Staff present were Val Braun, Steve Carter, Gary Caruthers, and Jerry Slaughter.

Expiration of 1979 Resolutions: By-laws changes adopted in 1979 are to remain in effect. All other resolutions will be allowed to expire unless reconsidered in May.

New Resolutions: The following were reviewed:

- Medicare DRG
- Long Term Care
- Local Health Departments
- Hospital Medical Staff Section
- Nuclear War Preparedness & Civilian Military Contingency Hospital System
- Professional Liability Tort Reform
- Control of Pediculosis — Dermatology Section

- KMS-UKSM Liaison Committee
- Others to be submitted:
 - Outpatient Clinics
 - Judicial Committee Peer Review
 - Administrative By-Laws Changes
 - Interim Legislative Study
 - Chiropractors — Sedgwick County

KaMPAC Board: Mrs. Mary Boyd was appointed to replace Dot Meyer, who has resigned to seek a seat in the Kansas Legislature.

Policy Determination: The Council considered a request by the Medical Society of Sedgwick County for a policy determination with respect to the role of the local county health department on the Primary Care Network program. It was agreed that all primary care should be delivered by the Primary Care Network physician in accordance with the contract.

Legislative Update was given by Jerry Slaughter.

AMA Voluntary Fee Freeze: The Council endorsed the concept of the proposal. Considerable emphasis was given to the physician-patient relationship when determining medical fees.

Annual Meeting Update: Staff reviewed the plans for the 1984 Annual Meeting May 3-6 at the Hutchinson Holidome, and the 1985 Annual Meeting May 2-5 at the Broadmoor in Colorado Springs. The 1988 meetings will be held in the Greater Kansas City Area.

See the July issue
of THE JOURNAL
for actions of the
House of Delegates



10 ways

to tell if you need a CPA.

- ☐ **1** Are your tax returns getting complicated? (Perhaps because family earnings are now from *several* sources or because your business is growing or changing?)
- ☐ **2** Do you need *immediate* tax write-offs through proper tax planning?
- ☐ **3** Would you consider changing some business procedures to cut *future* taxes?
- ☐ **4** Are you setting up or changing estate plans for yourself or someone else?
- ☐ **5** Is your business required to file financial reports — anything from a statement of earnings to full-scale audited financial statements?
- ☐ **6** Do you feel it might be time to incorporate your business?
- ☐ **7** Are you preparing a business loan application — and need supporting documentation?
- ☐ **8** Do you want to set up or improve a bookkeeping system?
- ☐ **9** Are you thinking of starting a pension or profit-sharing plan?
- ☐ **10** Do you need an *independent* source of professional management advice on how to use your resources more efficiently or how to make financial projections?

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ROSTER

The 1984 Roster will appear in the August issue of THE JOURNAL. NOW is the time to make sure that the information is accurate and up to date. Please examine your listing in the 1983 roster and advise us of any corrections we need to make.

If you are unable to locate your 1983 roster, contact our office (1-800-332-0156) and we will check the information for you.

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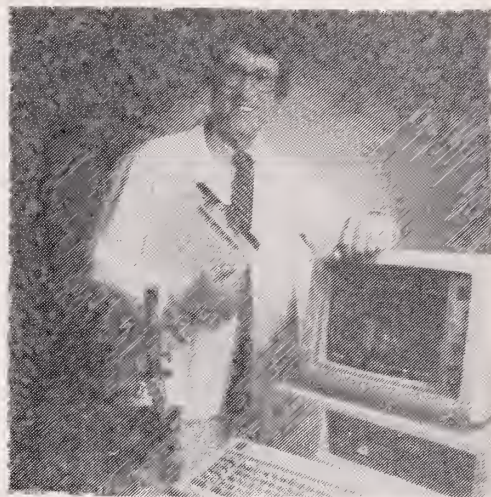
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Pancreatic Cancer

(Continued from page 140)

tases were found, although local invasion was present in UKSM patients.

Islet cell adenoma occurred as a higher percentage of benign neoplasms at UKSM than at MSKCC.

Acknowledgements

Frederick F. Holmes, M.D., Director, Tumor Registry, and staff assisted with the study. Indexes, reports, and histologic sections and tissue of many pancreatic cancers were made available for examination by Fritz Lin, M.D., Chief, Division of Surgical Pathology. T. Tomita, M.D. (UKSM) supplied data for islet cell tumors. Helen Darling and Ella Olson, Autopsy Service Division, UKSM, assisted in the retrieval of cases from autopsy files.

References are available from Dr. Waxman, UKSM-KC, 39th & Rainbow Blvd., Kansas City KS 66103.

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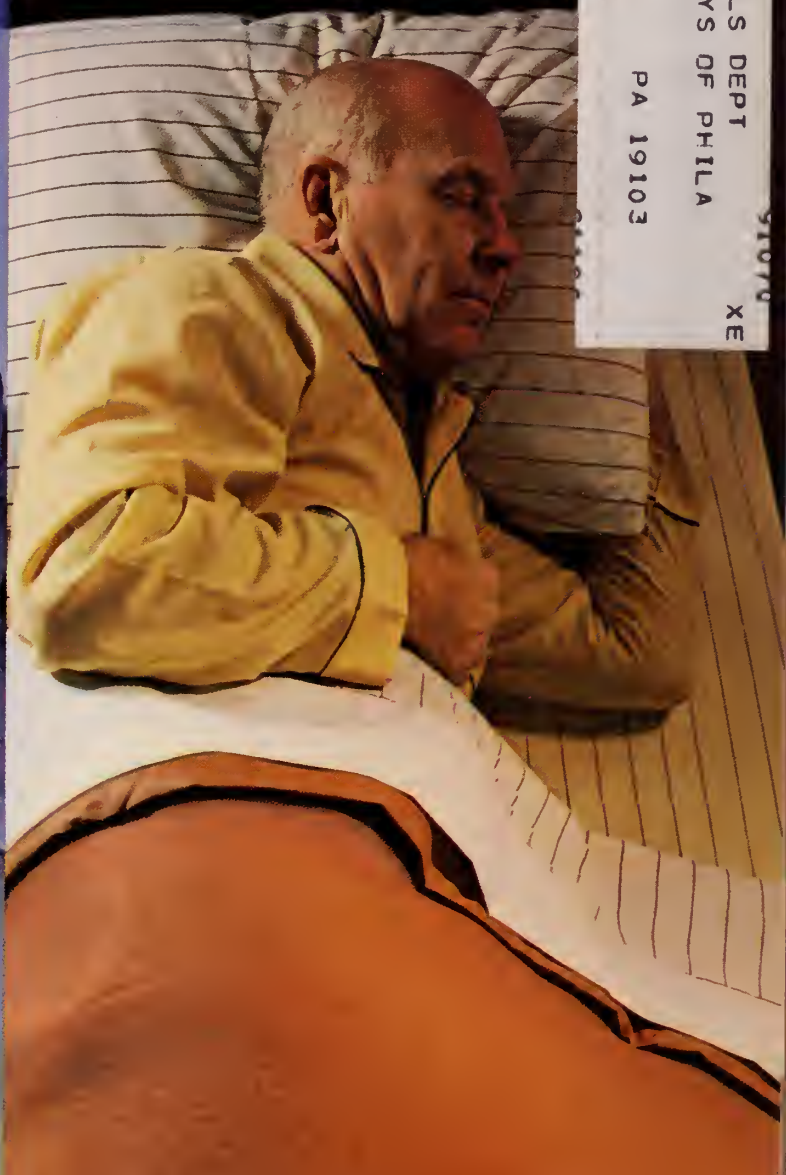


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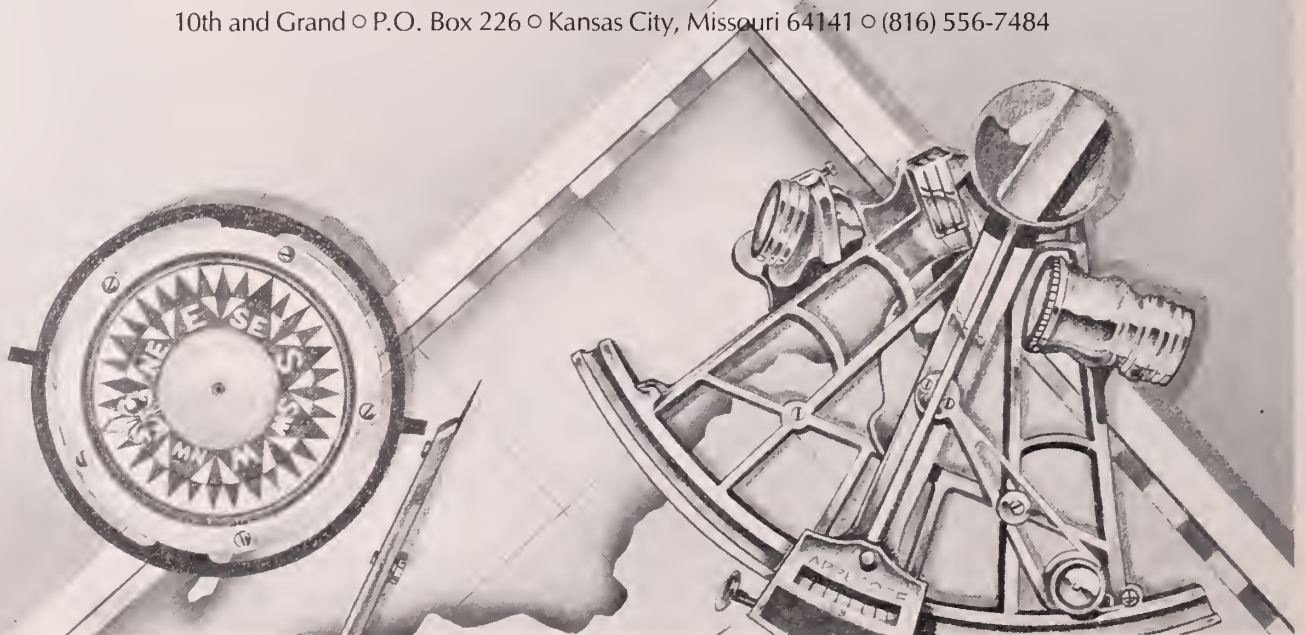
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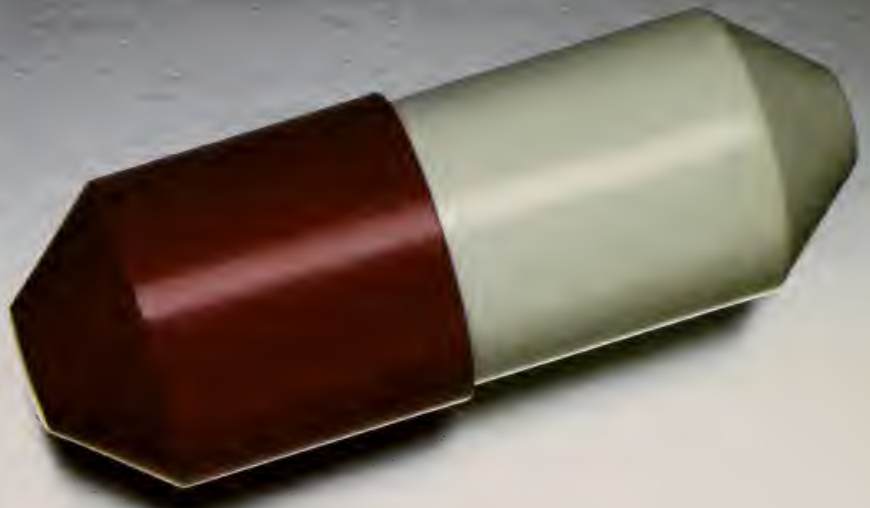
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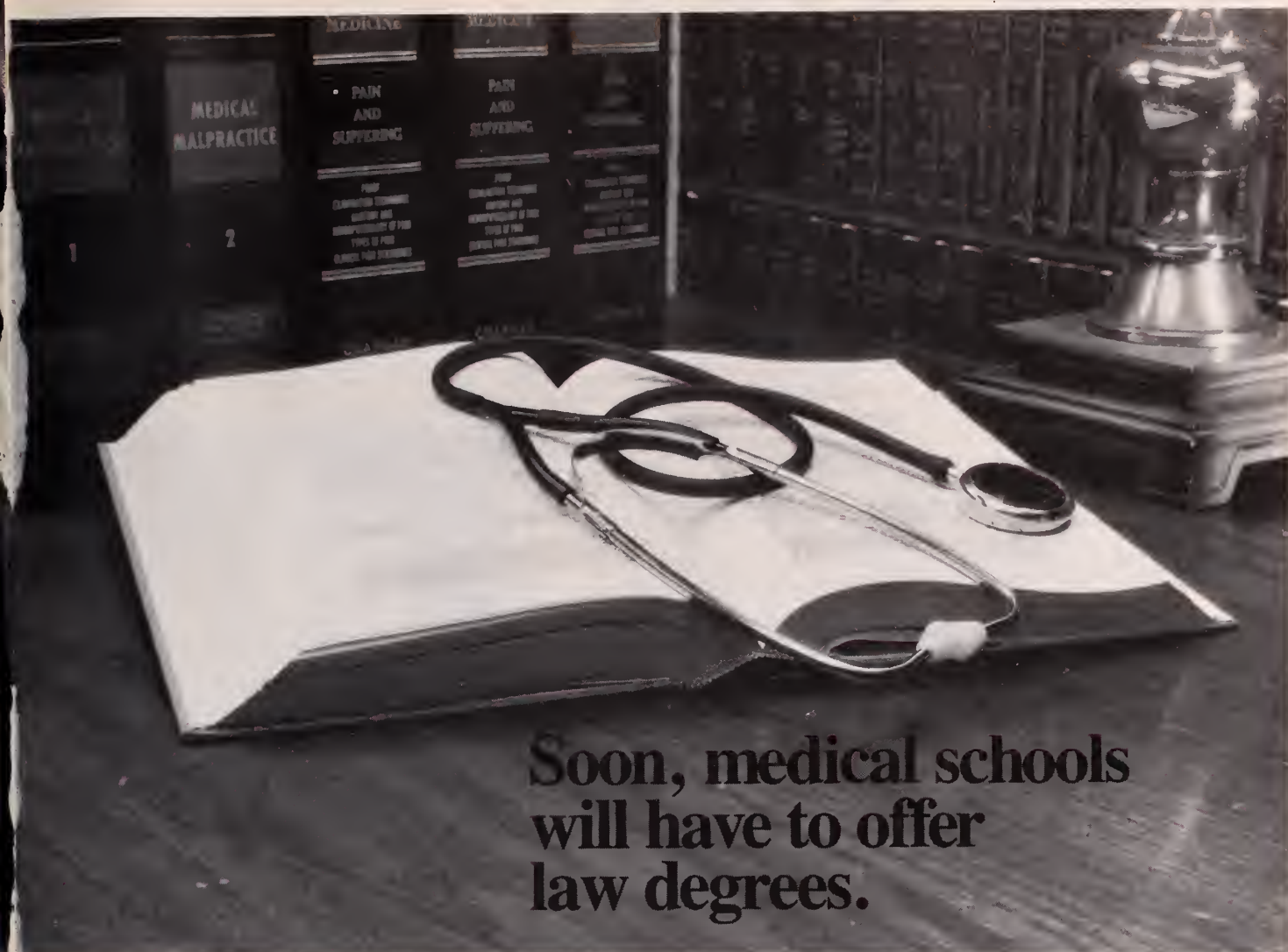
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Physiological Cardiac Pacing

ALFRED M. TOCKER, M.D.; LILIA RODRIGUEZ-TOCKER, M.D.;
ERNEST R. RODRIGUEZ, M.D. and JAPHET G. JOSEPH, M.D., *Wichita*

CARDIAC PACING is a dynamic medical discipline that reflects the technological advances of the present age. The single chamber pacemaker has been well-accepted and proven over many years, and is indicated over all other pacing modalities in selected cases. Ventricular single chamber pacing, however, has potential disadvantages that include loss of atrial contribution to ventricular filling, atrioventricular valve regurgitation, loss of normal sequence of ventricular activation, and decrease in vascular resistance by a vagally mediated atrial reflex, with resultant hypotension. It has been suggested that VVI pacemakers be implanted only in those patients who have high degree A-V block in the presence of atrial fibrillation or atrial flutter, or occasionally, prophylactically, in patients who have had rare episodes of transient complete block.

The advantages of synchronous pacing were recognized shortly after the development of the early cardiac pacemaker.¹⁻⁴ More recent developments of physiologic pacing systems offer significant advantages to those who may qualify for implantation of these units, estimated to be 15 to 70 per cent⁵ or more of the pacemaker patient population. Some authorities feel all pacemaker patients should have the advantages of physiologic pacing unless contraindications exist to implantations of such units.^{6, 7} Physiologic cardiac pacing refers to the application of atrial and ventricular sequential contractions, thereby restoring A-V synchrony, promoting more effective mechanical and electrical performance of

the heart and, in some patients, restoring rate variation.

A code has been developed to identify the various pacing modalities. Initially, a three-position code — such as VVI — was used. With the development of the more complex pacemakers programmable and applicable to tachyarrhythmia treatment, this code has been expanded to a five-position code (*Table I, Figure 1*).

Major limiting factors that retard the growth of physiologic pacing have been the unreliability of atrial electrodes with regard to sensing and position stability. However, atrial appendage ("J") leads have enjoyed successful clinical application. Tined, cork-screw, and pincher leads have been developed (*Figure 2*). Anatomical studies have shown that the available lead designs are well suited for the dimensions and features of the right atrium, and that a suitable atrial appendage is available in the majority of patients even after amputation during open heart surgery.^{5, 8} Epicardial leads, of course, may also be used.

Strong accusations that many physicians are unnecessarily implanting pacemakers have received national publicity, although investigations by accredited and responsible medical commissions have disproved such accusations. The joint American College of Cardiology/American Heart Association Ad Hoc Task Force on Assessment of Cardiovascular Procedures (Subcommittee on Pacemaker Implantation) has issued a set of "Guidelines for Permanent Cardiac Pacemaker Implantation." Also, the Health Care Financing Administration for Medicare has issued conditions and limitations with reference to payment for implantation of pacemak-

Address reprint requests to Japhet G. Joseph, M.D., Wichita Cardiac Clinic, 1111 No. St. Francis, Wichita KS 67214.

TABLE I
PACEMAKER CODES

CHAMBER PACED

- A = atrium
- V = ventricle
- D = dual (that is, both atrium and ventricle)

CHAMBER SENSED

- A = atrium
- V = ventricle
- D = dual
- O = none

MODE OF RESPONSE

- I = inhibited
- T = triggered
- D = dual (two modes of response; *i.e.*, atrial inhibited and ventricular triggered)

PROGRAMMABILITY FEATURES

- P = programmability of rate and/or output.
- M = multiprogrammability of rate, output, sensitivity, mode etc.
- O = none

TACHYARRHYTHMIA FUNCTION

- B = burst stimuli
- N = normal rate competition
- S = single- or double-timed stimuli
- E = externally controlled

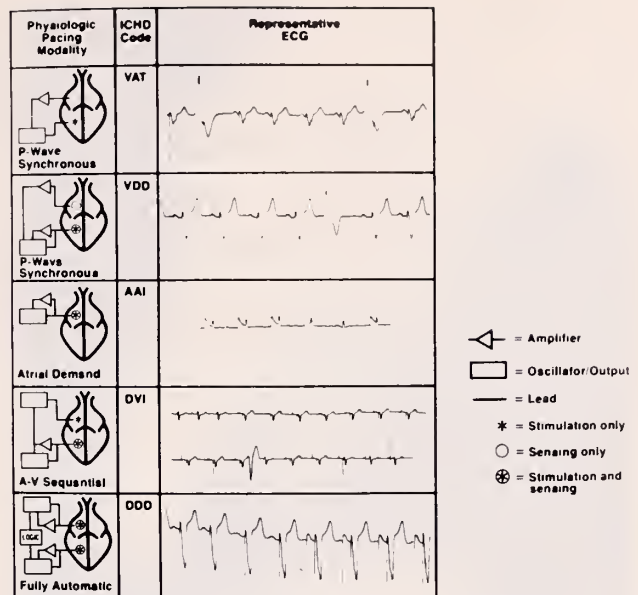


Figure 1. The various pacing modalities and representative ECGs. (With permission from Zipes, D. P., *Current Clinical Applications of Dual Chamber Pacing*. Medtronic, 1982)

ers. At this time, in most hospitals, each and every pacemaker implantation is reviewed to determine if the procedure is appropriate.

AAI Pacemaker

The single chamber atrial (AAI) pacemaker is not strictly a physiologic pacemaker because it is not rate responsive. It is indicated in cases of sick sinus syndrome, bradycardia syndrome and certain atrial or ventricular arrhythmias. An intact normal A-V node and conduction system are required, and it is contraindicated in the presence of chronic atrial fibrillation or flutter, bifascicular block, when there is evidence of an electrically silent atrium with complete or partial atrial paralysis, and carotid sinus syndrome. Indications for this pacemaker are becoming fewer and fewer. Many of those in whom the atrial (AAI) pacemakers were initially implanted have now proved to be unsuitable candidates for atrial pacing. For example, the incidence of A-V block increased in patients with sick sinus syndrome, and there also was an increased incidence of carotid sinus hypersensitivity.

DVI Pacemaker

Although the DVI pacemaker has no special requirements, it has certain limitations and disadvantages. This A-V sequential pacemaker senses in the

ventricle and then paces the atrium and ventricle synchronously based on the ventricular rate, but it does not allow for an increased heart rate with exercise. Indications include sick sinus syndrome, bradycardia syndrome, second or third degree A-V block, A-V reentrant tachycardias, and ectopic atrial or ventricular arrhythmias. Contraindications are chronic atrial fibrillation and flutter, and electrically silent atrium. The DVI pacemaker could potentially compete with atrial activity, which could conceivably produce atrial arrhythmias.

VDD and VAT Pacemakers

The VDD and VAT pacemakers also have certain limitations and disadvantages. This type of pacemaker is indicated in the presence of A-V block with normal sinoatrial node function. As this pacemaker modality paces the ventricle at the patient's own sinus rate, it allows the heart rate to increase with exercise. However, a significant limitation exists in that when atrial activity falls below the lower rate setting of the VDD pacemaker, it behaves as a VVI unit, thus creating the potential for A-V dissociation. It is contraindicated in the presence of chronic atrial fibrillation or flutter and an electrically silent atrium. Requirements include absence of atrial tachyarrhythmias and retrograde (V-A) conduction, satisfactory atrial signal amplitude and, for the VAT mode only, infrequent PVCs.

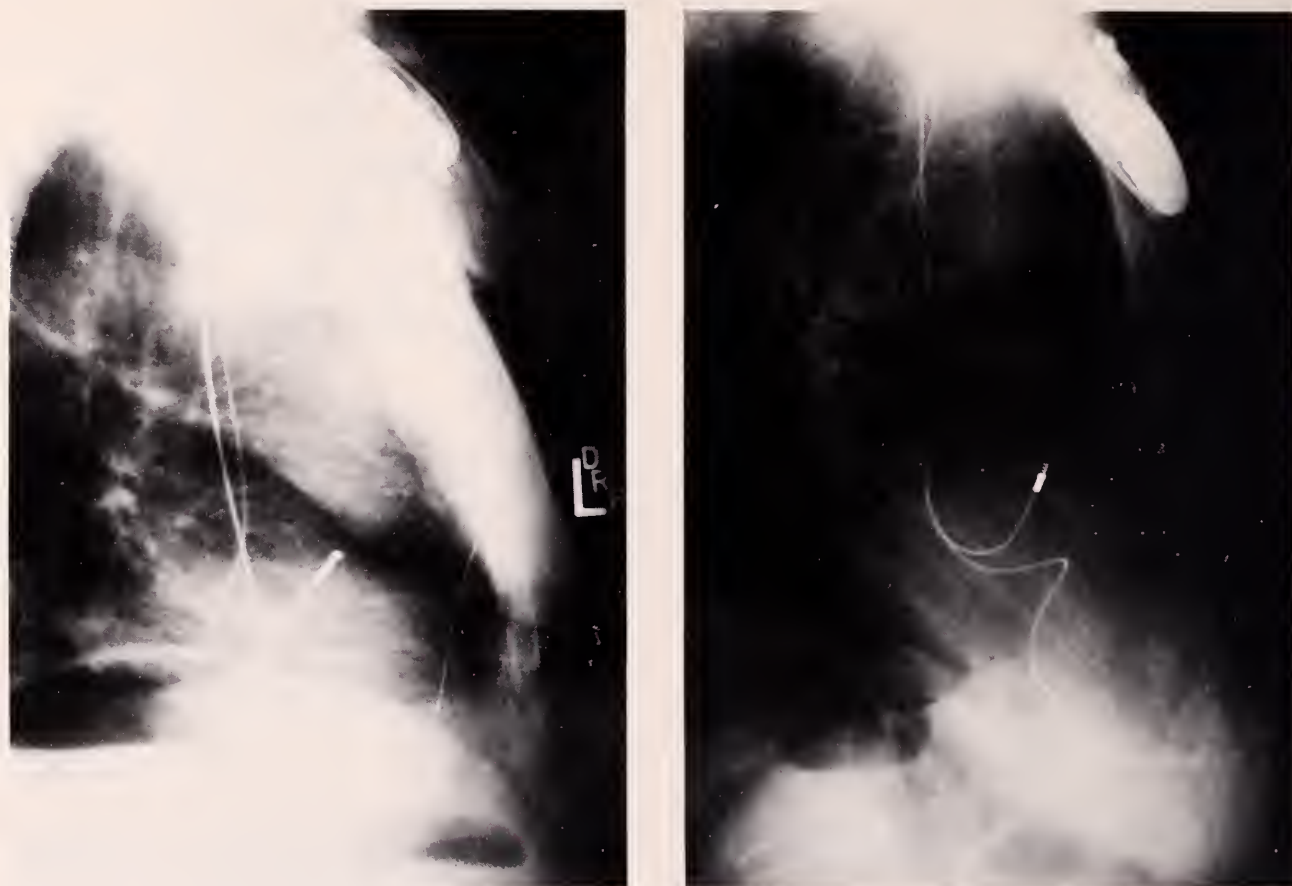


Figure 2. Atrial "J" leads. A. Tined B. Screw-in (Also shown are ventricular leads.)

Fully Automatic DDD Cardiac Pacemaker

Predicted to become the most accepted pacemaker modality, the newest unit is the DDD pacemaker, which combines properties of the DVI and the VDD units. DDD pacing not only offers the hemodynamic advantages of properly timed atrial systole, but it does so in more clinical situations than any other mode of pacing. The variety of programmable features in one such unit is set forth in *Table II*, demonstrating multiprogrammability of seven pacing parameters and five modes, including two dual-chamber modes, a special atrial refractory period that minimizes chances of pacemaker-mediated tachycardia, improved upper rate behavior that maintains cardiac output when atrial rates exceed programmed upper rates, and ventricular safety pacing for prevention of pacemaker self-inhibition. The fully automatic DDD cardiac pacemaker ensures atrioventricular synchrony, which minimizes or eliminates episodes of reentrant tachyarrhythmias. Multiprogrammability in DDD pacing optimizes clinical flexibility to meet the changing needs of the pacer patient.

Initially considered a pacemaker for implantation in the young and active patient, the DDD pacemaker is now considered for patients who lead an active lifestyle regardless of age. As of July 18, 1983, the Cordis Corporation reported the median age of the patients in whom the Sequicor DDD pacemaker had been implanted was 71 years. This represented a period of approximately two years in which 52.6 per cent had been implanted in patients 71 years of age or older, 27 per cent in patients between the ages of 61 and 71 years, 11.4 per cent in those between the ages of 51 and 60 years, 7.3 per cent in patients between the ages of 21 and 50 years, and only 0.9 per cent in those 20 years of age or younger.

Indications for implantation of the DDD cardiac pacemaker are listed in *Table III*. The best candidates for implantation of the DDD system are those who have a normal functioning sinus node with some type of A-V disturbance. Contraindications for implantation of the DDD pacemaker system include atrial fibrillation or flutter and significant atrial tachycardia.

Maintaining the DDD modality or reprogramming to another modality is determined primarily by

TABLE II
PROGRAMMABLE PARAMETERS*

Mode	A-V Universal (DDD)	A-V Sequential (DVI)	Ventricular Demand (VVI)
Lower Rate	40-80 ppm (increments of 10 ppm)	40-120 ppm (increments of 10 ppm)	40-130 ppm (increments of 10 ppm)
A-V Interval	25-250 ms ^{2, 3} (increments of 25 ms)	25-250 ms ⁴ (increments of 25 ms)	NA
Upper Rate ^{2, 3}	100, 125, 150, 175 ppm	NA	NA
Atrial Pulse Width	0.05, .1, .2, . . . 1.5 ms	0.05, .1, .2, . . . 1.5 ms	NA
Ventricular Pulse Width	0.05, .1, .2, . . . 1.5 ms	0.05, .1, .2, . . . 1.5 ms	0.05, .1, .2, . . . 1.5 ms
Atrial Sensitivity ⁵	0.75, 1.5, 3.0 mV Asynchronous	NA	NA
Ventricular Sensitivity ⁶	2.5, 5.0 mV Asynchronous	2.5, 5.0 mV Asynchronous	2.5, 5.0 mV Asynchronous

*Medtronic Versatrax II Model 7000A Universal A-V Pacemaker.

the atrial rhythm, which also determines the modality to which the system is to be reprogrammed. Reprogramming from the DDD to another mode is indicated with the appearance of sustained pacemaker-mediated tachycardia, profound bradycardia where rate responsiveness is of no benefit, and loss of atrial sensing.

Also of importance is the ability of these complex units to be monitored to solve many pacing problems, including problems to which the system itself gives rise. Until the advent of pacemakers that sense in both the atrium and ventricle, the problem of production of pacemaker-mediated tachycardia had not existed. The tachycardia is caused by retrograde conduction following a ventricular contraction, either spontaneous or caused by pacemaker stimulus. The dual chamber sensing pacemaker senses the retrograde P and conducts it antegrade to trigger another pacemaker stimulus, continuing the tachycardia. This tachycardia, with the pacemaker as the antegrade limb, continues in an "endless loop" until terminated by loss of atrial sensing or fatigue of the retrograde conduction limb, or until it is ended, in some way, by the pacemaker.

Future Cardiac Pacemakers

Further improvements in cardiac pacemakers, leads, and programmers are expected. At this time, clinical investigations continue to be carried out in the United States and elsewhere on technically ad-

vanced cardiac pacemakers. These implantable pacemakers, in addition to including several multi-model and multiprogrammable features, have models capable of tachycardia detection and treatment
(Continued on page 175)

TABLE III
INDICATIONS FOR FULLY AUTOMATIC DDD
CARDIAC PACERS

Acceptable

Requirement for AV synchrony over a wide range or rates such as:

- The active or young patient with atrial rates responsive to clinical need.
- Significant hemodynamic need.
- Pacemaker syndrome during previous pacemaker experience, or a reduction in systolic blood pressure of more than 20 mm Hg during ventricular pacing at the time of pacemaker implantation.

Marginal

- Complete heart block or sick sinus syndrome and stable atrial rates.
- Any patient where simultaneous control of atrial and ventricular rates inhibits tachyarrhythmias or in whom pacemaker can be adjusted to a mode designed to interrupt the arrhythmia.

Unacceptable

- Frequent or persistent supraventricular arrhythmias.
- Ineffective atrial contractions.

Percutaneous Drainage of Renal Abscess

KANG-NING HU, M.D.; ASSEFA WOLDESEMAIT, M.D. and
MURALIDHARA G. RAO, M.D., *Leavenworth*

RENAL ABSCESS is a rare disease with high mortality and morbidity. Thorley *et al.*,¹ in 1974, reported that 50 per cent of patients with renal abscess eventually die of the disease, and in one-third the condition is not diagnosed until surgery. In 1980, Anderson and McAninch² reported a mortality rate of 33 per cent for patients with renal abscess.

Despite such improved diagnostic methods as radioisotope scanning, computed tomography and angiography, development of better antibiotics and advancements in surgical techniques, the mortality and morbidity rates have changed little in the past 50 years.³

Case Report

A 30-year-old man was admitted because of chills and fever of two days' duration. In 1975, he had sustained a gunshot injury to the anterior chest, resulting in spinal cord injury at the level of T7 and paraplegia. He was able to perform intermittent self-catheterization after his recovery from the spinal shock, and the urinary tract was free of infection for several years.

One year prior to admission, he moved into a nursing home. The intermittent catheterization program was discontinued and a long-term Foley catheter was placed. Subsequently, he had several episodes of urinary infection, and had been hospitalized for treatment of *Pseudomonas* urinary infection and septicemia two months prior to this admission.

On admission, the patient's temperature was 40.1C; pulse, 120/min; and blood pressure, 80/40 mm Hg. Findings on chest examination were essentially normal. Since the patient is a T7 paraplegic, sensation below the chest could not be determined. No abdominal mass was felt. Urinalysis showed 10-25 WBC/hp, and WBC was 15,000/cmm. Urine culture grew *E. coli* and enterococcus. Blood culture showed *E. coli*. He was immediately treated with intravenous ampicillin and gentamycin. Subsequent blood culture showed anaerobic organisms: *Peptococcus prevotii* and *Peptococcus saccharolyticus*. Although bacteriologic studies indicated that the

organisms cultured were sensitive to the antibiotics administered, the patient still had a fever of about 37.8-38.9C intermittently for 12 days.

A gallium scan disclosed marked gallium accumulation in the left kidney. A sonogram revealed renal abscess involving the upper two-thirds of the left kidney. Intravenous pyelography also showed a large abscess at the upper pole of the left kidney, with displacement and distortion of the upper calyces and pelvis. A CT scan showed the abscess located at the anterolateral aspect of the upper pole of the left kidney (*Figure 1*).

Percutaneous aspiration of the renal abscess was performed. A 22-gauge needle was used to puncture the abscess cavity, and grey purulent material was aspirated. An 18-gauge needle was then inserted into the abscess cavity and a 0.008 inch guide wire was introduced into the abscess through the lumen of the needle. Both needles were then removed, and a 12F J-tip catheter was inserted into the abscess cavity along the guide wire after the percutaneous tract had been dilated with fascial dilators.

About 20 ml of purulent material was then easily drained from the catheter. A small amount of contrast medium was injected into the abscess cavity and x-ray revealed a multi-loculated abscess within the irregular contour of the cavity wall (*Figure 2*). The abscess cavity was gently irrigated with one-quarter-strength povidone-iodine solution that same



Figure 1. CT scan shows the abscess located at the anterolateral aspect of the upper pole of the left kidney.

From the Veterans Administration Medical Center, Leavenworth.

Address reprint requests to Dr. Hu, Chief, Section of Urology, Department of Surgery, Veterans Administration Medical Center, Leavenworth KS 66048.



Figure 2. X-ray following contrast injection reveals a multiloculated abscess.

day and on the following days. Bacteriologic culture showed mixed aerobic (*E. coli*, enterococcus, *Proteum mirabilis*, and *Providencia stuartii*) and anaerobic (*Bacteroides Melaninogenicus* and *Peptococcus saccharolyticus*) organisms. The patient became afebrile by the next day and has been well since then. The drainage fluid gradually became clear and scanty. The percutaneous catheter was removed after three weeks, and a followup CT scan showed complete resolution of the abscess after three months (Figure 3).

Discussion

Early manifestations of renal abscess are constitutional and nonspecific. General malaise, fatigue, anorexia, weight loss, intermittent fever and chills, abdominal pain or flank pain, and leukocytosis may be present. Because the most common cause of renal abscess is known to result from urinary tract infection, urine bacteriologic studies are very important in helping to make the diagnosis and provide a useful guideline for the administration of antibiotics. Frequently the differential diagnosis of renal abscess from acute pyelonephritis or acute focal nephritis is difficult in the early stage. Patients with acute pyelonephritis or acute focal bacterial nephritis usually respond to antibiotics within 48 hours. For those patients who do not respond to antibiotics, a high index of suspicion for the diagnosis of renal



Figure 3. Followup CT scan at three months shows complete resolution of the abscess.

abscess is warranted. With usage of intravenous pyelography and sonography, a presumptive diagnosis of renal abscess is usually made. CT scan is useful in questionable cases and in planning drainage procedure. Gallium scan and angiography are rarely useful. The diagnosis of renal abscess is eventually confirmed by either percutaneous aspiration or surgical exploration. However, percutaneous aspiration is quicker and safer than open procedure.

If purulent materials are discovered during the needle aspiration, a percutaneous catheter can be placed into the abscess cavity at the same time. Placement of a percutaneous catheter could provide sufficient and continuous drainage, allow irrigation or instillation of antibiotic solution topically, and can prevent reaccumulation of purulent materials.

In contrast to the conventional open drainage procedure, the percutaneous catheter drainage technique has special advantages for patients in poor condition and not suitable for general anesthesia. In 1977, Malgieri *et al.*¹⁴ reported a mortality rate of 7 per cent and a morbidity rate of 60 per cent (which mainly consisted of pulmonary complications and persistent cutaneous fistula) in patients who underwent the open drainage procedure. These complications have not been found in patients treated with percutaneous drainage procedure. It is suggested that the percutaneous catheter drainage should be the first step in treatment of renal abscess other than administration of antibiotics. In patients who do not respond to the percutaneous catheter drainage procedure, conventional open drainage procedure then is required. Most of these patients have abscess with multilocular cavities or a severely infected and damaged kidney which is not salvagable. Nephrectomy is usually the outcome in these patients.

References are available from Dr. Hu, Veterans Administration Medical Center, Leavenworth KS 66048.

Ventricular Septal Defect

KENNETH W. HOLLIS, M.D.*; JAMES P. BYRNE, M.D.† and
DAN A. FRANCISCO, M.D.,‡ *Wichita*

NON-PENETRATING thoracic injuries most commonly result from motor vehicle accidents.¹ Cardiac injury is being recognized with increasing frequency. Whereas this injury has been associated with a high mortality rate, improved emergency communication and transportation systems have improved survival in these victims. Penetrating cardiac injuries usually produce overt symptomatology and are easily recognized. However, blunt cardiac damage, which is more common, is subtle and may be easily missed.

Cardiac injury is now considered the most frequent unsuspected visceral injury responsible for fatality from accidents.² These injuries may be categorized as involving the pericardium, myocardium, valves, or coronary arteries (*Table 1*).

Myocardial contusion occurs more frequently than has been recognized.^{2, 3} Its incidence ranges from 25 per cent of all blunt chest injuries to as high as 76 per cent.² Although myocardial contusion usually does not result in death, it may be associated with potentially lethal complications.⁴

This report presents a case of myocardial contusion and the resulting complication of ventricular septal defect.

Case Report

A 14-year-old white male was involved in a motorcycle accident. Upon deceleration, he was thrown from the cycle and was struck in the precordium by one of the handlebars. Initial care consisted of routine stabilization and observation. At that time, there were no observed cardiopulmonary problems.

Approximately 24 hours later he was noted to have developed a loud holosystolic murmur over the precordium with a palpable thrill. Congestive heart failure developed. This prompted emergency transfer to the cardiovascular service at Wesley Medical Center, Wichita.

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Presented at the Annual Meeting of the American College of Surgeons, Kansas Chapter, September 1983.

Address reprint requests to Dr. Hollis, Wesley Medical Center, 550 No. Hillside, Wichita KS 67214.

Physical examination revealed ecchymoses of the precordial area. A grade 6/6 holosystolic murmur was present as noted above. The patient was obviously dyspneic, and mild peripheral edema was evident.

The chest roentgenogram showed bilateral pulmonary congestion and mild cardiomegaly. The electrocardiogram showed left axis deviation with non-diagnostic anterolateral ST depression, sinus tachycardia, and an intraventricular conduction delay. The left ventricular voltage was increased with inferior ST elevation. The creatinine was 2.3 mgm/dl.

Following admission, the patient underwent immediate right heart catheterization including an oximetry series. Based on the step-up in oxygen saturation within the right ventricle, the diagnosis of ventricular septal defect was made. A two-dimensional echocardiogram clearly demonstrated the location of the defect (*Figure 1*). A left ventriculogram performed one day later confirmed the diagnosis. A percutaneous intra-aortic balloon was placed and counterpulsation instituted. This produced stabilization of the patient's condition. However, it became apparent that the patient was not going to improve enough to allow delayed closure of the defect. Therefore, 11 days after the injury, operation was performed. There was no visual evidence of myocardial contusion. Utilizing cardiopulmonary bypass, a

TABLE I
CLASSIFICATION OF BLUNT CARDIAC INJURIES³

Pericardium —
Disruption
Hemopericardium
Pericarditis
Myocardium —
Contusion
Rupture
Septal perforation
Late aneurysm
Valves, chordae tendinae, papillary muscle —
Rupture
Coronary arteries —
Contusion and thrombosis
Laceration

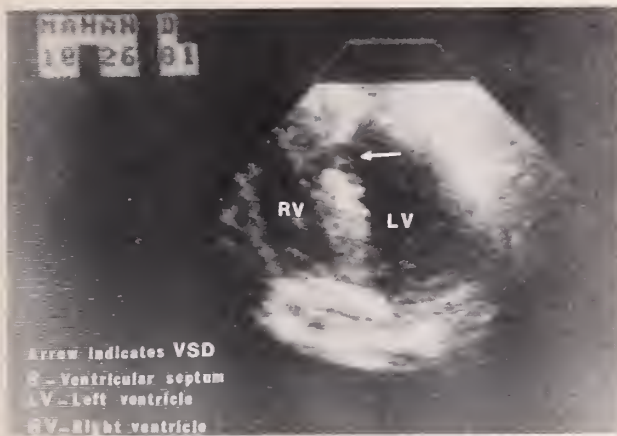


Figure 1. Real time echocardiogram showing VSD shortly after patient's arrival.

left ventriculotomy was done and the defect was closed with a Dacron patch. Following operation, the patient's cardiac status improved markedly. The intra-aortic balloon was removed and he was dismissed 3.5 weeks post-injury. Subsequent followup at frequent intervals during the past 24 months has indicated a full return to normal activity without evidence of sequelae. A two-dimensional echocardiogram done after surgery showed an intact ventricular septum (Figure 2).

Discussion

Rupture of the interventricular septum from blunt cardiac trauma is an unusual event. The mechanism of septal rupture appears to be compression of the heart between the sternum and spine which results in contusion with immediate or delayed rupture. The heart is most vulnerable during late diastole or early systole when the ventricular chambers are distended and the valves are closed.⁵

Swan-Ganz cardiac catheterization for diagnosis of this entity has been previously employed.⁶ The production of a left-to-right shunt and resultant increase in oxygen saturation of blood sampled from the right ventricle as compared to the right atrium allows the diagnosis to be made. The use of two-dimensional echocardiography has not been previously reported as an adjunctive diagnostic modality in the evaluation of cardiac trauma. Because of the ease of performance and its non-invasive nature, we propose that this method be employed early in the course of these patients. These two bedside procedures simplify and expedite the diagnostic evaluation of these patients.

It has traditionally been recommended that repair be delayed until the heart has recovered from the associated contusion low cardiac output state. Additionally, repair is technically easier with delay be-



Figure 2. Echocardiogram showing surgically placed dacron graft successfully repairing VSD.

cause fibrosis along the periphery of the defect allows more secure suture placement.⁶ While we were unable to delay repair for six weeks to allow some healing of the defect, use of the intra-aortic balloon did allow recovery from the myocardial contusion.

References are available from Dr. Hollis, Wesley Medical Center, 550 No. Hillside, Wichita KS 67214.

Vox Dox

Vox Dox Editor:

A statement in my article, "Hormonal Patterns in Menstrual Dysfunction" (*The Journal*, April 1984, page 123, right hand column, line 30), has been brought to my attention: "If a progestin or spironolactone is prescribed on a regular basis, the patient must be advised of possible risks or teratogenicity if pregnancy results concomitant to the use of these medications."

While there is some data linking the use of oral progestin during pregnancy with teratogenicity, there is none with the use of spironolactone, according to Michael S. Anderson, M.D., of G. D. Searle & Company (personal communication). Nor has there been a report of feminization of a male fetus in a woman using spironolactone during pregnancy. Nevertheless, G. D. Searle & Company does not recommend the use of spironolactone during pregnancy.

The statement in the article was based on information contained in the *Physicians' Desk Reference* and was submitted prior to my receipt of the letter from the Searle representative.

PAUL REITH, M.D.
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The President's Message

The actions of the House of Delegates are being analyzed and plans are being made to implement those directives. May's annual meeting in Hutchinson had the largest attendance of any in history. Thirty-six resolutions were introduced. Discussion was lively. A summary of proceedings, including the resolutions as passed, will be published in the July issue of the *Journal*. One can readily see by reading these resolutions that physicians in Kansas have many concerns today.

In the President's remarks to the House of Delegates, I listed my priorities for the coming year as: (1) Membership; (2) Medical liability; (3) Development of young leadership; (4) Interaction with governmental agencies; (5) Physician-hospital relations; and (6) Communications.

The first priority is strengthening the membership. We now have the most members in the entire history of the Kansas Medical Society. Yet there are more practicing physicians in Kansas who are not members of the Medical Society than at any time in the past. My goal is for every practicing physician in Kansas to be a member of the Medical Society. I ask each of you who knows a non-member physician to please contact him or her. Outline the many advantages of membership. Encourage enrollment so that the added voice can further strengthen our Society.

What are the advantages of being a member of the Kansas Medical Society? As stated in the Constitution of this organization: "The object of this Society is to unite the medical profession of the State of Kansas in promoting the science and art of medicine and protecting the health of the citizens of this State."

Through numbers there is strength. Your individual voice of concern is magnified by the impact of others across the state. State legislation and the regulations of state agencies shape our daily practice of medicine. The individual physician depends upon



the state medical society for information, interpretation, and help for positive change in dealing with statewide issues.

Activities of the Medical Society include: (1) Liaison with governmental agencies, state medical school, and voluntary health care organizations; (2) Educational activities for physicians through the *Journal* and the scientific program at the annual meeting, summary position papers such as "Kansas Reporting Laws" and "Preventing Medical Malpractice," the accreditation of CME Category I institutions across the state other than medical school accreditation; (3) Various forms of patient education; (4) Information for education of the general public; and (5) Practice management aids.

Limitations of time and space preclude further description of beneficial activities of the Medical Society. I encourage you to visit the Topeka office of your Society and find out more of the Society activities. The building is on the southeast corner of Topeka Avenue and 13th Street, just four blocks south of the Capitol complex.

Finally, the August issue of *The Journal of the Kansas Medical Society* is the membership directory of the KMS. Simply being listed in that directory is worth the entire annual membership dues. Please notify the office of any corrections in your listing prior to July 1.

F. Calvin Buehler, MD
President



Shopper's Service

Although the pattern has been inexact and never completely uniform, fashions in medical professional housing have been, over the years, a reflection of the social currents not fully appreciated. Early in the century when Manifest Destiny was still operative and physicians enjoyed greater autonomy than at any time since, the business community became identifiable as "Downtown." Physicians whose offices had more often been located in their homes or adjacent buildings began to move to individual buildings there — over the bank or drug store with their presence announced to the citizenry by the gold leaf legends in the windows on the Main Street side.

As Downtown grew outward, buildings grew upward. Physicians began occupying them and increasingly sacrificed autonomy to share space and sometimes professional responsibilities with their colleagues. Hospitals, since they often grew from residences, were located away from Downtown but, since they housed the seriously or chronically ill, more of the physician's time was spent in the office or patient's home. With the surgeon in the ascendency and increasing sophistication of patient care in general, hospitals came to occupy more of the physician's attention. Downtown was becoming congested, parking was an increasing problem and, with the automobile as midwife, urban sprawl was born although not immediately recognized as a monster by its parents. Physicians increasingly moved to the neighborhood of the hospitals in medically oriented buildings, even extending their professional attachments at times to the previously abhorred group practice (whatever that says about autonomy).

Hospitals, increasingly frustrated by lack of consistent and dependable emergency room coverage, began hiring physicians to provide it, adding to the already hospital-housed pathologists, radiologists, and anesthesiologists.

Downtown was suffering more than the loss of physicians. Shopping centers were developing at the community periphery and it was discovered that by putting a roof over them and calling them malls, customers would walk miles through them although they complained if they couldn't park within half a

block of a downtown destination. Malls represented urban sprawl in its post-adolescent form, reaching that state of being on a diet of customers sautéed in credit cards, seasoned with self-service, and strained through check-out lines.

Meantime, the public was being assaulted through all of its senses by promotion of substance and service of every variety. An air of impatience was prevalent — why wait and save for the car, TV, swimming pool, and so on when easy payments made them immediately available? The medical mystique fell on hard times because of the increasing public conviction that it was inaccessible, was less than perfect in each of its efforts, and cost too much anyway. Consequently, physicians found they were spending much of their time accommodating philosophy, method, and delivery to the merchandising spirit of the day. It was probably inevitable, then, that medical services should come to occupy cubicles in the malls, claiming convenience for their customers and, presumably, economy. Nor did hospitals, increasingly alert to ways of extending their image and purpose throughout the community, overlook this opportunity and sponsored some, thereby assuring more direct sustenance for their diminishing censuses.

Medical offices have of late sought a sort of anonymity — no mention of the contents in individual or personal terms but rather using some medically-oriented designation, usually ending in P.A. Since an essential for a mall installation is a distinctive name, whether bestowed by the franchiser or created locally, the opportunities for medical groups are numerous. Traditionalists will have to guard against seeing anything sinister in the trend since each of the past moves has brought its critics, but disgruntled colleagues on the outside might favor a pertinent designation such as "The Body Snatchers." "Health Merchants, Inc." comes to mind but may be a little too prosaic. Since primary care is to be emphasized, "First Aides" might appeal to some. Then there is a certain catchiness to "Mall-practice Unlimited" but on second thought that isn't too good. But in keeping with the spirit of the thing, we can expect a prize contest to find the best designation and you are free to make up your own list of prizes.

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An added complication... in the treatment of bacterial bronchitis*

Increasing incidence
of ampicillin resistance in
Haemophilus influenzae

Ampicillin-Resistant
Haemophilus influenzae

H. influenzae

S. pneumoniae

Brief Summary Consult the package literature for prescribing information.

Indications and Usage: Cefclor® (cefclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms.

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae* (*Diplococcus pneumoniae*), *Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci).

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Cefclor.

Contraindication: Cefclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Cefclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins), therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out.

Precautions: General Precautions—If an allergic reaction to Cefclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of Cefclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Cefclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Cefclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinistix® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in terrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Cefclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers—Small amounts of Cefclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one

Some ampicillin-resistant strains of *Haemophilus influenzae*—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Cefclor.¹⁻⁶

In clinical trials, patients with bacterial bronchitis due to susceptible strains of *Streptococcus pneumoniae*, *H. influenzae*, *S. pyogenes* (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Cefclor.⁷

Cefclor®

cefclor

Pulvules®, 250 and 500 mg

hour. The effect on nursing infants is not known. Caution should be exercised when Cefclor® (cefclor, Lilly) is administered to a nursing woman.

Usage in Children—Safety and effectiveness of this product for use in infants less than one month of age have not been established.

Adverse Reactions: Adverse effects considered related to therapy with Cefclor are uncommon and are listed below.

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthritis and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Cefclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations of SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

(061782R)

* Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.

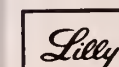
Note: Cefclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

References

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Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285. Eli Lilly Industries, Inc., Carolina, Puerto Rico 00630.

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Cardiac Pacing

(Continued from page 163)

functions (suppression capabilities) which will offer new versatility in patient management and will be capable of telemetering atrial or ventricular intracardiac electrocardiograms for valuable troubleshooting information. Under clinical investigation are models that will offer the first DDD pacing in a bipolar configuration which many have shown to be superior to unipolar in preventing myopotential sensing and inhibition.⁹⁻¹³ Improvement in leads is expected to significantly reduce pacing thresholds and improve sensing with a potential for greatly improving the patient safety factor, with reduced energy requirements and increased pacemaker longevity.¹⁴ An endocardial, steroid-eluting, pacing lead has been developed to treat chronic high thresholds and exit block (*i.e.*, the inability to elicit cardiac depolarization from the energy in a standard pacemaker stimulus).¹⁵⁻¹⁷ Programmers are expected to be more versatile, simplifying and extending the programming functions.

Automatic Implantable Defibrillator

The automatic implantable defibrillator is undergoing clinical trials in the United States.^{18, 19} The device is a self-contained unit designed to detect

malignant ventricular arrhythmias as they occur and to terminate them with a defibrillating current. Early clinical trials demonstrate its safety and effectiveness in terminating ventricular tachyarrhythmias as well as its ability to reduce markedly the incidence of sudden cardiac death in high-risk patients.

The most recent unit, called AID-B defibrillator, consists of a unit in an electronically sealed pack similar in size to the early pacemakers. This unit is fairly bulky because of the large batteries required for cardioversion or defibrillation pulses, the latest units being able to develop an output of up to 42 joules. The unit requires three leads: one unipolar lead placed in the superior vena cava; another bipolar lead placed in the right ventricle (for accurate determination of the lower heart rate); and a left ventricular epicardial lead with a large surface pad. The superior vena cava-left ventricular apex lead circuits are used for detecting ventricular tachycardia and for delivery of the corrective pulses. The superior vena cava lead and the right ventricular bipolar lead are both inserted transvenously but the epicardial lead pad is inserted via a small thoracotomy.

In the very near future, indications for the clinical use of these units will be further delineated, and very soon, more sophisticated units will be available for general use.

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CHALLENGES FOR CLINICAL NUTRITION IN THE 80s: 1984 postgraduate course program, Sept. 10-11, 1984, Marriott Pavilion Hotel, St. Louis MO. For information, contact American Society for Parenteral & Enteral Nutrition (A.S.P.E.N.), 1025 Vermont Avenue, NW, Suite 810, Washington DC, 20005; 202-638-5881.

8th SUMMER INSTITUTE FOR DRUG DEPENDENCE: August 26-31, 1984, Antlers Plaza Hotel, Colorado Springs CO. For information, contact The Institute for Integral Development, P.O. Box 2172, Colorado Springs CO 80901; 303-634-7943.

Virginia Heart Institute provides on-site consultation for administrators interested in development of ambulatory facilities including outpatient cardiac catheterization. If interested, write Patricia Ferree, Virginia Heart Institute, 205 N. Hamilton St., Richmond VA 23221.

Summary

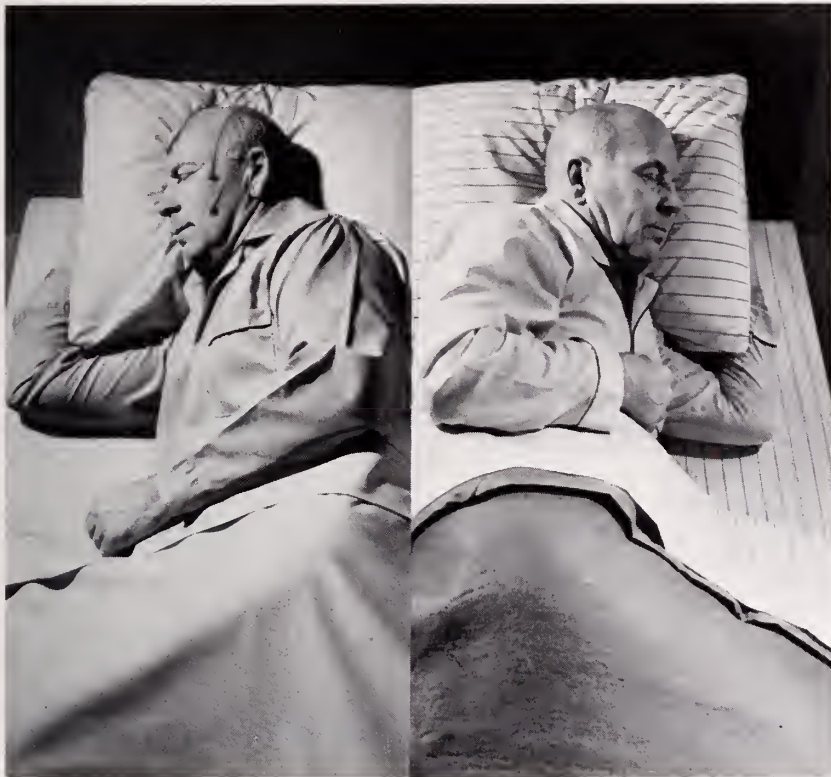
Physiologic pacing systems respond to the needs of circulation under all conditions by restoring normal activation sequence of the heart (atrioventricular synchrony) and the ability of the heart to alter its rate physiologically. These pacemakers are indicated as a replacement unit in those patients with implanted univentricular pacemakers suffering from "Pacemaker Syndrome," and in those patients who cannot afford the hemodynamic, electrophysiologic, or metabolic penalty associated with the univentricular pacing systems.

Of the physiologic pacemakers, the multiprogrammable DDD units now existent and being developed are, by their versatility, the ones of choice for those patients who require physiologic pacing. The increased cost of these units has to be balanced against the probable benefits to the patient when considering their implantation.

At the present time, clinical investigations are underway in the development of improved pacemaker pulse generators, leads, and programmers which will result in more efficient pacemaker function and which will also offer detection and treatment of tachycardia problems. Further improvements in pacemaker pulse generators, leads, and programmers are to be expected in the future.

References are available from Dr. Joseph, Wichita Cardiac Clinic, 1111 No. St. Francis, Wichita KS 67214.

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Caution patients about driving, operating hazardous machinery or drinking alcohol during therapy. Limit dose to 15 mg in elderly or debilitated patients. Contraindicated during pregnancy.

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References: 1. Kales J et al: *Clin Pharmacol Ther* 12:691-697, Jul-Aug 1971. 2. Kales A et al: *Clin Pharmacol Ther* 18:356-363, Sep 1975. 3. Kales A et al: *Clin Pharmacol Ther* 19:576-583, May 1976. 4. Kales A et al: *Clin Pharmacol Ther* 32:781-788, Dec 1982. 5. Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc* 27:541-546, Dec 1979. 6. Kales A, Kales JD: *J Clin Pharmacol* 3:140-150, Apr 1983. 7. Greenblatt DJ, Allen MD, Shader RI: *Clin Pharmacol Ther* 21:355-361, Mar 1977. 8. Zimmerman AM: *Curr Ther Res* 13:18-22, Jan 1971. 9. Amrein R et al: *Drugs Exp Clin Res* 9(1):85-99, 1983. 10. Monti JM: *Methods Find Exp Clin Pharmacol* 3:303-326, May 1981. 11. Greenblatt DJ et al: *Sleep* 5(Suppl 1):S18-S27, 1982. 12. Kales A et al: *Pharmacology* 26:121-137, 1983.

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Contraindications: Known hypersensitivity to flurazepam HCl; pregnancy. Benzodiazepines may cause fetal damage when administered during pregnancy. Several studies suggest an increased risk of congenital malformations associated with benzodiazepine use during the first trimester. Warn patients of the potential risks to the fetus should the possibility of becoming pregnant exist while receiving flurazepam. Instruct patient to discontinue drug prior to becoming pregnant. Consider the possibility of pregnancy prior to instituting therapy.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, light-headedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase, and paradoxical reactions, e.g., excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg recommended initially until response is determined.

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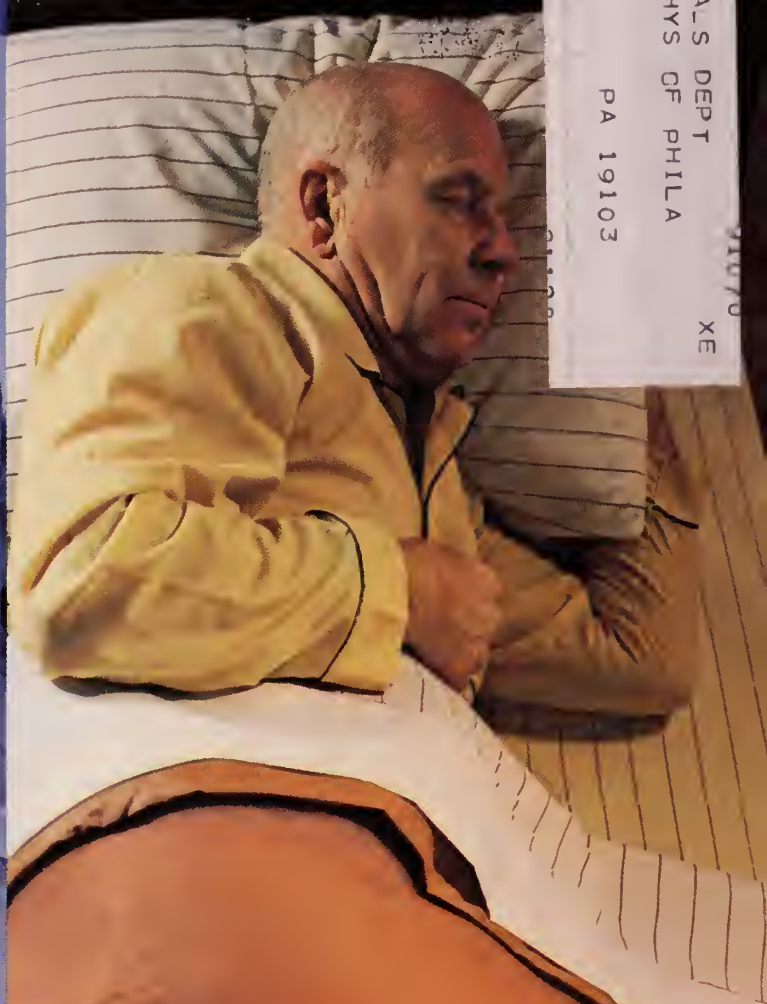
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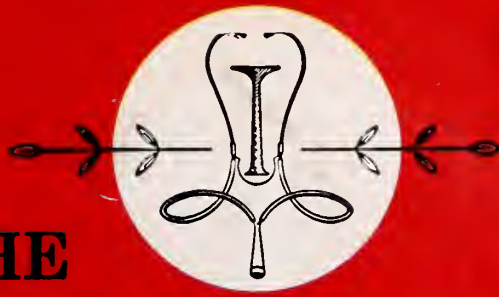
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The JOURNAL of the KANSAS MEDICAL SOCIETY

Contents for July

Special Articles

Handicapped Children Referral in Kansas — Mary Mira, Ph.D., Kansas City, Kansas, and Karl Altman, Ph.D., Lawrence

The primary care physician is usually the first to recognize a child as handicapped and is the crucial link between the family and services. The earlier the intervention, the greater the benefits 193

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Official Proceedings

1984 Annual Meeting of the House of Delegates

Transactions of the 125th Annual Session of the Kansas Medical Society are published in this issue of *The Journal*.

The Resolutions are printed in numerical order under the Minutes of the Second House of Delegates Session. Those Resolutions that were not adopted but were referred for further study or information are so indicated. The Resolutions that failed to pass are retained in the Official Minutes at the Executive Office, but are not reported here.

FIRST SESSION

The First Session of the House of Delegates of the Kansas Medical Society met on Saturday, May 5, 1984, beginning at 9:00 AM at the Holiday Inn Holidome, Hutchinson. The meeting was called to order by G. Rex Stone, M.D., Speaker. Dr. Stone briefly reviewed the order of business for the day and explained that the House was composed of elected officers, past presidents, representatives of specialty societies, and the duly elected delegates from the component medical societies. He stated that the House would follow the *Sturgis Standard Code of Parliamentary Procedure*.

Dr. Stone then introduced Donald R. Brada, M.D., President of the Reno County Medical Society, who welcomed Delegates and KMS and Auxiliary members to Hutchinson.

Dr. Stone announced appointment of tellers as follows: Rex R. Fischer, M.D., Manhattan; Virginia T. Gruendel, M.D., Kansas City; and Erwin T. Janssen, M.D., Topeka.

The Speaker announced the presence of the official quorum. The minutes of the preceding meeting were approved.

Primary election ballots were distributed. There being no additional nominations from the floor, the members voted for the following slate:

PRESIDENT ELECT: Clair C. Conard, M.D., Dodge City

FIRST VICE PRESIDENT: Franklin G. Bichlmeier, M.D., Kansas City

SECOND VICE PRESIDENT: Robert J. Haskins, M.D., Chanute; Donald W. Hatton, M.D., Lawrence; Wayne O. Wallace, Jr., M.D., Atchison

CONSTITUTIONAL SECRETARY: Erwin T. Janssen, M.D., Topeka; Richard M. Skibba, M.D., Wichita

TREASURER: James A. Loeffler, M.D., Wichita; Richard Meidinger, M.D., Topeka; Roger D. Warren, M.D., Hanover

SPEAKER: G. Rex Stone, M.D., Manhattan

VICE SPEAKER: Herbert Fransen, M.D., Newton; Edwin D. Rathbun, M.D., Liberal

AMA DELEGATES, 1985-86: Alex Scott, M.D., Junction City; Kermit G. Wedel, M.D., Minneapolis

AMA ALTERNATES, 1985-86: Warren E. Meyer, M.D., Wichita; Linda D. Warren, M.D., Hanover

David E. Gray, M.D., presented the Necrology Report, as follows:

It has been recorded that only since about 2500 B.C. has mankind recognized the inevitability of death. It should be added that since that time and probably before, physicians have pursued their continuing but futile effort to refute it. Perhaps this is why the death of a physician has been accorded a special meaning and adds to the poignancy of this moment in our organizational year when we acknowledge the deaths of our colleagues.

Name and address	Age	Date
E. L. Andre Baude, M.D., Topeka	81	1/4/84
Edgar H. Beahm, M.D., Independence	70	12/30/83
Laurie J. Benz, M.D., Wichita	44	6/19/83
Victor E. Bolton, M.D., Kansas City	59	10/16/83
Gilbert N. Casady, M.D., Hutchinson	60	10/11/83
Stephen S. Ellis, M.D., Coffeyville	70	11/2/82
Joseph E. Gootee, M.D., Topeka	62	11/16/83
Roy D. Grayson, M.D., Albuquerque, New Mexico	92	7/14/83
Robert A. Haines, M.D., Topeka	66	9/20/83
A. Brooks Harrison, M.D., Wichita	82	6/12/81
Mary T. Hastings, M.D., Shawnee Mission	38	12/9/83
Cline D. Hensley, Jr., M.D., Wichita	62	5/7/83
Glenn H. Jackman, M.D., Estes Park, Colorado	77	7/30/82
Catherine M. Jackson, M.D., Wichita	43	10/16/83
Herman F. Janzen, M.D., Moundridge	91	4/6/83
Charles R. Kempthorne, M.D., Manhattan	80	5/11/83
Harry Lazar, M.D., Wichita	75	3/9/84
Lawrence R. Leigh, M.D., Shawnee Mission	71	6/12/83
R. Bruce McVay, M.D., Clay Center	86	7/20/83
Clyde W. Miller, M.D., Wichita	73	9/25/83
J. Russell Nevitt, M.D., Moran	83	5/1/83
Philip C. Nohe, M.D., Kansas City	66	10/17/83
Leonard A. O'Donnell, Sr., M.D., Wichita	78	12/18/82
Robert H. Riedel, M.D., Topeka	78	10/13/83

Anthony F. Rossitto, M.D. San Francisco, Calif.	72	8/11/83
Alfred S. Steinzeig, M.D., Shawnee Mission	70	11/8/83
William C. Swisher, M.D., Wichita	61	6/17/83
Karl A. Youngstrom, M.D., Kansas City	74	8/17/83

At the conclusion of the report, the House stood for a moment of silence in tribute to the members who departed during the past year.

Dr. Gray then presented the report of the Editorial Board:

With somewhat mixed feelings, I bring you an accounting of the Editorial Board's year. As Mr. Dickens noted of an earlier revolutionary period, it was the best of times, it was the worst of times. This can be transliterated to the modern idiom as an expression of the good news-bad news phenomenon. The subject is, of course, the status of the *Journal* and the bad news is our now-well-publicized financial embarrassment that has come to fruition since my last report. The good news is that our abbreviated format did produce, for the moment at least, an abundance of material for publication. This has required some extra hours of work in revising and reducing, not to mention the impossible task of devising a diplomatic approach to rejection. Let me hasten to add, however, that nothing in these remarks is to be interpreted as discouragement to prospective contributors. Don't hesitate to keep us busy — just keep them short.

I can report that our struggle toward a life of economic virtue has been getting an A for effort but so far no encouragement to return to our prodigal ways so perhaps, to paraphrase Mr. Dickens' ending to that tale, it is a far, far better thing we do than we have ever done. But we acknowledge that the guillotine is still in working condition so our continued efforts will determine whether it is a far, far better rest we go to than we have ever known.

A prime factor in our current condition, apart from the lack of business acuity on the part of the Board, continues to be the progressive loss of pharmaceutical advertising. As you are probably aware, many public watchdogs are certain that you have been corrupted by the pharmaceutical manufacturers' considerable influence through support of your medical periodicals. Meantime, the manufacturers are certain they can do better with their advertising dollars than bestow them on state journals, hence the loss of a prime source of revenue. We have been gratified, however, to note an increase in local advertising from both medically-related and lay business sources. It will do no harm for those of you with prospects for such among your friends to ac-

quaint them with this means of reaching the medical population of the state or let those already using the *Journal* columns know of our appreciation.

It has always been an important feature of my reports to recognize the loyal and valued efforts of Val Braun and Eleanor Bell in bringing the *Journal* into being since without them it literally — and literarily — could not exist. During the past year more than ever, however, they have gone above and beyond the call of duty and the Board is more than ordinarily indebted to them.

Recognizing that he has been the firm but kindly intermediary with the Council in assessing our situation and guiding us in the ways of economic righteousness, we have resisted any kill-the-messenger impulses toward the Business Manager. We do deny, moreover, that the frustrations of dealing with the Editorial Board are the main factor in his decision to renounce the world in favor of a life of contemplation and prayer in ivy-covered halls. We thank him for these years of assistance, wish him good luck, and trust that the ivy will not be of the *Rhus radicans* variety.

At the same time, we are pleased to note the revival of a hit show from past years, "Slaughter on Topeka Avenue." We welcome Jerry back to the fold although the calf may be a little too skinny to butcher yet. Economy being the watch word, we may still have some stationery around with his name on it.

Despite the austerity of our publication life, we do retain the right to one mild extravagance, the annual bestowing upon our incumbent president of a bound volume of the year's *Journals* to ease the prospect of his approaching unemployment. If nothing else in these days of high technology, it will, when properly applied, do an excellent job of eliminating ganglions. Mr. President, our best wishes.

Dr. Gray and Jimmie A. Gleason, M.D., retiring president, then exchanged bound volumes of *The Journal*.

The House heard the following oral reports:

Executive Director — Steve Carter

Treasurer — Wayne O. Wallace, Jr., M.D.

Legislative Committee — Erwin T. Janssen, M.D., Steve Carter

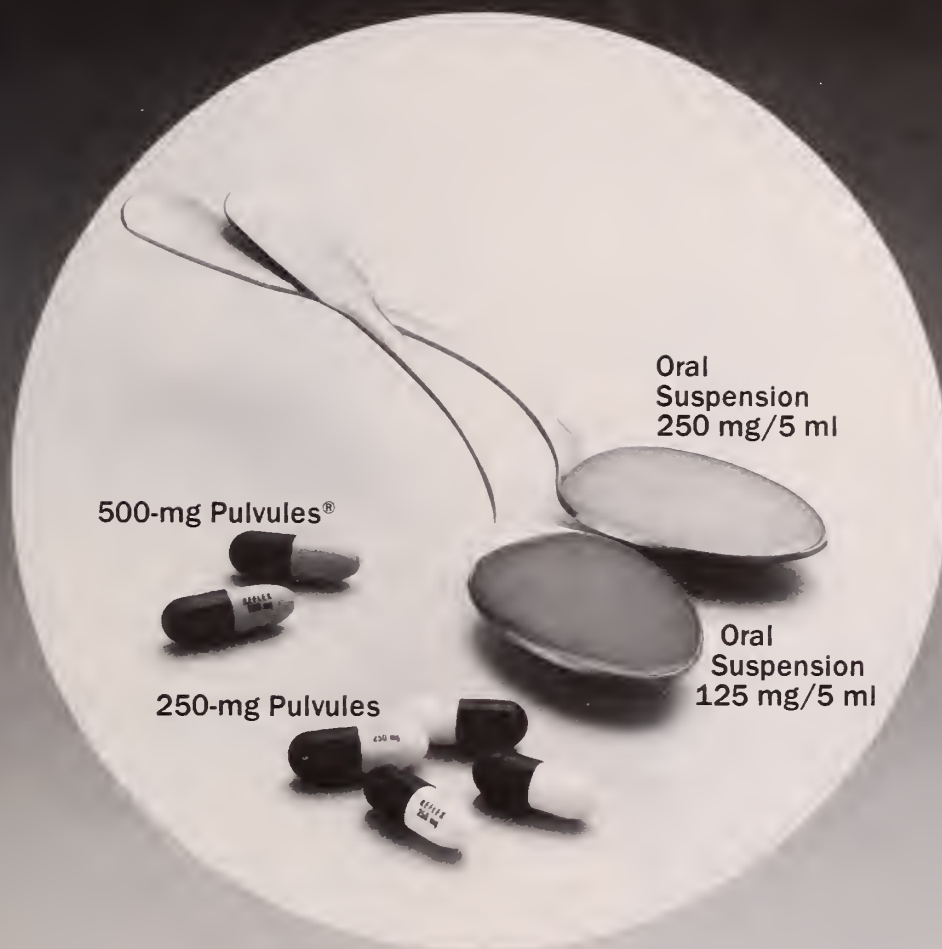
Continuing Medical Education — Donald W. Hatton, M.D.

Maternal Health — Rex R. Fischer, M.D.

Richard Meidinger, M.D., in addition to an oral report, presented the following written report from the Committee on Health and Environment Liaison:

This Committee was mandated by KMS Resolu-

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tion 83-4, to provide liaison with the Department; to identify problem areas which can mutually be considered, evaluated and addressed; and to identify and promote areas of mutual interest. The new Committee replaces four separate committees (School Health, Emergency Medical Services, Health Resources and Study of Health Departments).

The Committee:

- Identified a long list of mutual concerns, ranging from the activities of the local health departments, to radiation control in physicians' offices, to perinatal health.
- Surveyed KMS membership on priorities and issues for this Committee. The top five areas were: health care costs; access to health care; aging; long term care; KDHE communications.
- Developed a Joint Policy Statement on Long Term Care, concerning inpatient and home care, public education, and coordination of local community efforts to promote a unified program.
- Developed a method of communication between the various segments of the Department and KMS members.
- Kansas Hospital Rules and Regulations have not been revised since 1974. A timetable was established to begin the process of updating the Rules and Regs with completion targeted for Fall, 1984. In this connection, contacted all medical specialty sections in Kansas, all local medical societies and the applicable KMS committees for input.
- Met with representatives of Kansas Association of Local Health Departments to explore mutual concerns.
- Studied in depth the activities of local health departments and developed a response to Resolution 83-4.

Committee recommendations:

- Recommend passage of Resolution 84-25, Local Health Departments.
- Recommend passage of Resolution 84-26, Long Term Care.
- Recommend that the Kansas Medical Society maintain an ongoing liaison with KDHE.

Daniel K. Roberts, M.D., prepared the following written report of the Professional Liability Committee:

The primary charge of the Committee this year was to develop recommendations to ensure the solvency of the Health Care Stabilization Fund. A secondary charge was to recommend possible tort reform proposals addressing the malpractice situation.

At its first meeting, the Committee approved hiring an independent actuary to review data on closed claims in the Health Care Stabilization Fund. The Committee also determined to explore the possibilities of recommending professional management of the HCSF with physician overview and a mechanism for evaluating multiple claim providers.

After reviewing the report of the independent actuary, the Committee developed the following recommendations:

- Removal of the statutory \$10 million maximum balance in the HCSF;
- Removal of the maximum 65% surcharge to develop a surcharge proposal to allow the HCSF to become financially solvent on an accrual accounting basis over a five-year period;
- Retain the unlimited coverage feature of the Fund;
- Increase the primary coverage limits from \$100/300,000 to \$200/600,000.

The specific details of this legislative session on SB-507 Professional Liability have been reported. The bill passed, and was signed by the Governor with the inclusion of the majority of the Committee's recommendations.

Proponents of the bill included the Insurance Department, Kansas Hospital Association, Association of Osteopathic Medicine, Board of Healing Arts, Kansas Chiropractic Association, and the Independent Insurance Agents.

The Committee identified the following issues for further study:

- Investigate private insurance companies and types of coverage available to determine the feasibility of phasing out the HCSF.
- Study the issue of tail coverage for resident physicians leaving the state and retiring physicians, perhaps establishing a minimum number of years of participation in the HCSF before the coverage would be purchased by the Fund.

The Committee published and distributed a booklet entitled "Preventing Medical Malpractice, The Physician's Responsibility."

The issue of tort reform was reviewed by the Committee. After considering all the variables, the Committee determined that any substantive tort reform proposals should not be considered in 1984. The 1985 legislative session was considered more appropriate, thus allowing time for research and coalition building to support our positions.

The Committee has identified several areas of tort reform that should be addressed. The proposals are listed in Resolution No. 24 and will be considered by this House later this afternoon.

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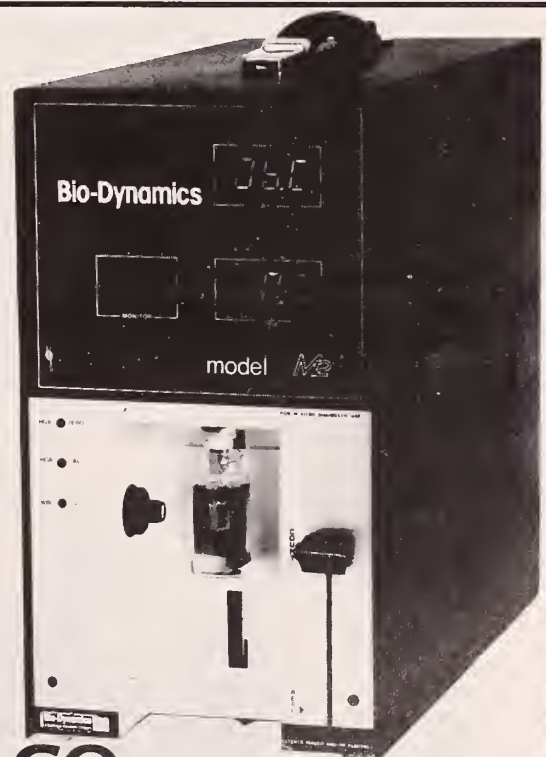
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The House then heard the following oral reports:
SRS Advisory Committee — Phillip A. Godwin, M.D.

KaMPAC — Roger D. Warren, M.D.

Kansas Foundation for Medical Care — Louis M. Culp, M.D.

AMA Trustee & Member, AMA Council on Long Range Planning & Development — W. J. Lewis, M.D., Dayton, Ohio

North Central Regional AMA Auxiliary Vice President — Betty Szewczyk (Edward), Belleville, Illinois

HCFA Advisory Committee — Kermit G. Wedel, M.D.

President's Report — Jimmie A. Gleason, M.D.

Alex Scott, M.D., submitted the following written report on the activities of the Committee on Aging:

- Developed the program for a series of Forums on Aging with the following participants:
 - Insurance Commissioner
 - Department on Aging
 - Department of Health/Environment
 - Medicare
 - Kansas Association of Broadcasters
 - KMS Auxiliary
 - Local Physician Representatives
 - Local Legislative Representatives

To date, the following Forums have been held: Emporia, Great Bend, Lawrence, Topeka, Wichita, Salina.

- In addition to holding regular committee meetings with the representatives of DOA, an open public meeting was held with the representatives of the various aging organizations and the public-at-large. A consumer survey questionnaire was distributed soliciting opinions on health care delivery to the aging population. The results indicated the following as the most prevalent concerns by the elderly:
 - Medicare assignments;
 - Health education — public not aware of ways to keep health care costs down;
 - Lack of information regarding prescription drugs;
 - How to monitor one's condition;
 - How to cope with a chronic disability.
- This committee developed a series of patient information brochures for distribution to the patients through the physicians' offices. These are available from the Executive Office at cost.
- Participated in a feasibility study to develop a continuing physician education on geriatrics.
- Made arrangements with the Kansas Silver-

Haired Legislature for physician coverage during that session in Topeka, November 13-16, 1984.

- This committee, in cooperation with the KMS Committee on Health/Environment Liaison, developed the statement on long term care which has been jointly adopted by the Kansas Medical Society, Kansas Department On Aging, Kansas Department of Health and Environment and Kansas Department of Social and Rehabilitation Services. This matter will appear before the House of Delegates in resolution form.
- Through the efforts of this committee, the Kansas Medical Society will be represented at the Governor's Annual Conference on Aging, May 15-17, 1984, in Pittsburg, through the presentations of the following programs:
 - "The Patient's Role in Reducing Costs"
 - "Senior Sexuality"
 - "When Enough Is Enough" — the cost of terminal illness

Current trends indicate that the elderly and the medical profession are moving toward serious reconciliation efforts. This change in attitude was quite evident in the experience of our committee. It is our firm conviction that better understanding of our mutual problems and concerns regarding health care delivery can be achieved through improved communication. This committee seriously recommends that physicians more actively participate at all levels of the debate with the aging population.

Robert J. Haskins, M.D., Chairman, presented the following report for the Allied Health Liaison Committee:

- The Committee interviewed the following groups:
 - Occupational Therapy Association
 - Kansas Athletic Trainer's Association
 - Kansas Respiratory Therapy Society
 - Kansas Society for Medical Technology
 - Kansas Dietetic Association
 - Kansas Academy of Physician's Assistants
- Reviewed the language in legislation pertaining to physical therapists: "Physical Therapists may evaluate patients without physician referral but may initiate treatment only after consultation with and approval by a physician licensed to practice medicine and surgery. . . ." The Committee regards this language as appropriate when considering applications by similar professions.
- Prepared a special issue of KMS Newsletter on Allied Health Personnel Credentialing.
- Submitted written testimony to the technical committees of Kansas Statewide Health Coordinating Council (SHCC) in support of registration for occupational therapists and respiratory therapists.



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- ☐ 4 Are you setting up or changing estate plans for yourself or someone else?
- ☐ 5 Is your business required to file financial reports — anything from a statement of earnings to full-scale audited financial statements?
- ☐ 6 Do you feel it might be time to incorporate your business?
- ☐ 7 Are you preparing a business loan application — and need supporting documentation?
- ☐ 8 Do you want to set up or improve a bookkeeping system?
- ☐ 9 Are you thinking of starting a pension or profit-sharing plan?
- ☐ 10 Do you need an *independent* source of professional management advice on how to use your resources more efficiently or how to make financial projections?

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The following actions were recommended:

- Approve registration for dieticians.
- The Committee recommends no change in the present status of Physician's Assistants.
- SHCC will be reassessing the credentialing process in Kansas. The Committee recommends that KMS continue to review credentialing applicants under the existing setup.

Diane Sanders (Alan), President of the KMS Auxiliary, distributed the following written report to the House:

"Reflecting on the year's activities, it became quite apparent to me that I am now basking in other people's glory. Perhaps I have finally made "cheerleader" — for a very prestigious team, the Kansas Medical Society Auxiliary.

As Ann Rempel, President-Elect, and I traveled around the state, we were quickly made aware that the county auxiliaries were already actively participating in the Auxiliary's projects. It was our happy duty to thank and give just praise for a job well done. No encouragement or goading was ever considered. What an easy and delightful task!

This year AMAERF, under the leadership of Nancy Macy, vigorously grew, with more counties having Christmas sharing cards, auctions, and other fund-raising events. Again, we will stage a gala evening's entertainment at the state meeting for AMAERF. This year AMAERF donations from Kansas amount to \$20,147.27 for unrestricted funds for UKSM at Kansas City, and \$5,262.22 for Wichita. The restricted funds (Medical Students Assistance Fund) included \$4,285 for Kansas City and \$245 for Wichita.

The Forums on Aging are another prime example of Auxiliary's involvement in a project that is of universal concern currently. The KMS Executive office, working with various state agencies, coordinated an excellent program which the following counties presented: Shawnee, Sedgwick, Barton, Flint Hills, Douglas, Saline, and later this month, Wyandotte. The good will and information disseminated on all levels through the state and local communities was a tremendous boost for medicine. Thanks to the physicians who participated and also to the state co-chairmen, Harriet Olson and Ruth Mitchell.

Another Auxiliary project which flourished this past year was legislation, with Carol Loeffler as chairman. Our LEGS Alert was very instrumental in getting SB-507, which concerned the confidentiality of peer review boards, passed. All Auxilians realize that we must be politically active.

The Auxiliary membership received letters recommending participation in the "Chemical People" program from Nancy Roderick, Drug Abuse Prevention Chairman. This was a marvelous tool to open the Pandora's Box of drug and alcohol abuse in our local teen-age populations. Our members saw the merits of the program and the response was excellent. Ongoing programs are blossoming all over the state. Last year the initial step was taken to implement the drug and alcohol abuse program for the Auxiliary and hopefully, this year is a bridge to even bigger gains for next year.

An extremely active state committee is International Health, with Dee Cauble at its helm and her basement as the hub of activity. This is truly a continuing network of Auxilians and interested Kansans. Dee placed a monetary value of over \$26,000 on items made and donated this past year by the Wichita-based group. Many other counties participated directly, or through Dee: Johnson, Barton, Harvey, Shawnee, Central Kansas, Saline, and Douglas.

Our membership is fast growing. Betty Glover, membership chairman, lists a total of 1,221 individuals who belong to the KMS Auxiliary, of which 114 are new members. This energetic and motivated membership is constantly responding to the needs of their individual communities and to medicine and health, in general."

The annual report of the Constitutional Secretary was presented as follows:

<i>Categories</i>	<i>1981</i>	<i>1982</i>	<i>1983</i>	<i>1984</i>
Active Members	2,146	2,225	2,218	2,288
First Year Active Members			16	18
Probationary Members	54	79	68	57
Associate Members	5	3	5	6
Intern/Resident Members	23	37	34	63
Delinquent Members	165	102	194	110
Dues Exempt:				
Emeritus Members	133	130	144	149
Honorary Members	1	1	1	1
LOA/Personal Exempt Members	40	24	26	28
Retired Members	165	167	200	209
Service Members	15	15	16	13
Students	4	8	38	345
TOTALS	2,751	2,791	2,960	3,287

Ivan E. Rhodes, M.D., Chairman of the Impaired Physicians Committee, presented the following written report:

- At the direction of the Council, because of legal requirements of SB-41, effective July 1, 1983, this Committee modified its operating policy. This was done to maintain confidentiality and to preserve the physician/patient relationship. We

have, therefore, not had access to individual cases of reported suspected impairment. However, some 25 new treatment or physician rehabilitation cases were handled by this Committee in 1983.

- Worked closely with various Senators and Representatives in 1984 to obtain passage of SB-507. This has now been signed by the Governor. This bill should help in maintaining confidentiality and further improve our efforts with the Board of Healing Arts.
- The Chairman served on the AMA Planning Committee for the Sixth National Conference on the Impaired Physician, to be held September 6-9, 1984, in Secaucus, New Jersey. The Chairman will also be a member of the faculty for this conference, at which some 400 physicians, medical students, licensing board personnel, and medical society staff from the U. S. and Canada are expected to attend.
- Developed with the KMS Council approval a policy of reimbursing committee members for expenses incurred when treating impaired physicians who have been referred other than through the Kansas Medical Society. All such referrals and specific arrangements for treatment will be decided between the referring source and the committee member.
- Recommend the continuation of this committee.
- Executive staff to prepare for Committee's use a clear outline of how SB-507 will affect the work of this body.

Ralph Baehr, M.D., Chairman of the Insurance/Membership Committee, presented the following written report:

- Dr. Linda Warren was appointed to chair membership recruitment activities with emphasis placed on the KMS established Resident Physician Section and Medical Student Section.
- The various insurance programs endorsed by the KMS have been reviewed. Alternatives for group health insurance are still under active investigation and our specific recommendations in this regard are anticipated subsequent to our next committee meeting.
- The AMA/Telenet was endorsed by our committee.
- Approved the addition of partial disability benefits to the Disability Income Program sponsored by Washington National Insurance Company.

The committee received the full support of the Society's staff. I would like to commend Gary Caruthers in particular for his thoroughness and persistence on behalf of the committee and Society.

Annual Meeting Grants

The Kansas Medical Society is grateful for the grants and contributions received from:

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Kansas City, Missouri

Franklin G. Bichlmeier, M.D., Chairman of the Judicial Committee, presented the following written report:

"The purpose of the committee is to actively work to assure that colleague physicians are of the highest character and professional ability; protecting patients from physicians who are deficient in moral character or professional competence; to assist component medical societies to establish due process mechanisms for handling complaints and to handle complaints that can't be resolved at the local level.

The Judicial Committee scheduled a meeting to review the effects of the new physician reporting law and to consider committee involvement in reviewing physician credentials and hospital staff privileges.

The passage of the physician reporting law places

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continued actions of the committee in possible jeopardy. The law requires any person licensed to practice the healing arts to report any knowledge which he possesses that another person has committed an act which is grounds for revocation, suspension or limitation of license. There is no specific obligation by the staff or the KMS to report complaints received until such knowledge is known by a licensee of the Board.

Legal Counsel continues to review this situation to determine its effects on the Committee.

The Committee recommends that the KMS pursue the peer review of members and nonmembers for problems of ethics or medical competence brought to our attention by trustees, medical staffs or individual physicians. Legal Counsel is currently drafting procedures to accomplish this."

The following Councilors or Alternates reported for their Council districts:

District #1 — Wayne O. Wallace, Jr., M.D., Atchison

District #2 — Louis M. Culp, M.D., Kansas City

District #3 — James J. Bridgens, M.D., Shawnee Mission

District #5 — Kenneth M. Boese, M.D., Manhattan

District #6 — James H. Ransom, M.D., Topeka

District #7 — John P. Brockhouse, M.D., Emporia

District #8 — Newton Smith, M.D., Arkansas City

District #10 — Richard Siemens, M.D., Lyons

District #11 — Clifton C. Schopf, M.D., Wichita

District #13 — Wallace N. Weber, M.D., Hays

District #14 — Wendale McAllaster, M.D., Great Bend

District #15 — Edwin D. Rathbun, M.D., Liberal

District #16 — John Neuenschwander, M.D., Hoxie

District #17 — Max E. Teare, M.D., Garden City

District #19 — Robert J. Haskins, M.D., Chanute

The following Council Districts were not officially represented at this session:

District #4 — George W. Pogson, M.D., COUNCILOR, Pittsburg; Rodney K. Odgers, M.D., ALTERNATE, Pittsburg

District #9 — Herbert D. Doubek, M.D., COUNCILOR, Belleville; Jack E. Lungstrum, M.D., ALTERNATE, Salina

District #12 — Carl D. Ambler, M.D., COUNCILOR, Pratt; Joel T. Weigand, M.D., ALTERNATE, Wellington

District #18 — David A. Leitch, M.D., COUNCILOR, Garnett; Stephen W. Myrick, M.D., ALTERNATE, Lawrence

All reports were unanimously accepted.

The House then adjourned for lunch and an address by Senator Nancy Landon Kassebaum.

Rex G. Stone, M.D., Speaker, reconvened the House at 1:00 PM. In addition to the 29 submitted in advance, the following resolutions were introduced:

Norman K. Pullman, M.D., Wichita: #30 — Pre-admission Hospital Certification; #31 — Medicare Exit Reviews; #32 — Physician Reimbursement by Third Party Payers

James H. Ransom, M.D., Topeka: #33 — Freedom of Choice of Primary Care Physician

Daniel N. Pauls, M.D., Parsons: #34 — Required Affidavit on Medicare Patients

Ronald W. Quenzer, M.D., Pratt: #35 — Preferred Insurance Service

The Speaker announced primary election results as follows:

Retained on the ballot:

SECOND VICE PRESIDENT: Donald W. Hatton, M.D., Lawrence; Wayne O. Wallace, Jr., M.D., Atchison

TREASURER: James A. Loeffler, M.D., Wichita; Roger D. Warren, M.D., Hanover

Dr. Stone announced that Council Districts 1, 3, 5, 8, and 9 need to elect new councilors and alternates. He then announced the Reference Committee members: Richard M. Skibba, M.D., Wichita, Chairman; Donald D. Moeller, M.D., Kansas City; Larry Rotert, M.D., Topeka; and Don R. Tillotson, M.D., Ulysses.

Following announcements of subsequent activities, the Speaker adjourned the First Session of the House of Delegates at 3:00 PM.

SECOND SESSION

The Second Session of the House of Delegates was called to order by the Speaker, G. Rex Stone, M.D., on Sunday, May 6, 1984, at 9:00 AM at the Holiday Inn Holidome, Hutchinson.

The Speaker announced the presence of a quorum and noted that the session would be conducted in accordance with *Sturgis Standard Code of Parliamentary Procedure*. Dr. Stone made some announcements and reviewed some rules. Each delegate was to be allowed an opportunity to be heard on every question, but except for the person making the motion, delegates were asked to cooperate by speak-

ing only twice on a single question.

The Speaker appointed the following tellers: John P. Brockhouse, M.D., Emporia; Rex R. Fischer, M.D., Manhattan; and Virginia T. Gruendel, M.D., Kansas City.

The following election results were announced:

PRESIDENT: F. Calvin Bigler, M.D., Garden City

PRESIDENT ELECT: Clair C. Conard, M.D., Dodge City

FIRST VICE PRESIDENT: Franklin G. Bichlmeier, M.D., Kansas City

SECOND VICE PRESIDENT: Donald W. Hatton, M.D., Lawrence

CONSTITUTIONAL SECRETARY: Richard M. Skiba, M.D., Wichita

TREASURER: Roger D. Warren, M.D., Hanover

SPEAKER: G. Rex Stone, M.D., Manhattan

VICE SPEAKER: Herbert Fransen, M.D., Newton

AMA DELEGATES, 1985-86: Alex Scott, M.D., Junction City; Kermit G. Wedel, M.D., Minneapolis

AMA ALTERNATES, 1985-86: Warren E. Meyer, M.D., Wichita; Linda D. Warren, M.D., Hanover.

Dr. Stone recognized Vernon W. Filley, M.D., Pratt, for having recently been awarded an Honorary Degree in Science by the Pratt Community College in recognition of his service to the school. Dr. Filley

was also the winner of the drawing for a computer at the ComputerLand display at the Holidome during the meeting.

Dr. Stone commended Jack D. Walker, M.D., Kansas City, for becoming a candidate for the Kansas Senate.

Jack R. Cooper, M.D., Shawnee Mission, commended Dr. Stone for his work as Speaker and Dot Meyer (Warren), Wichita, for her candidacy for the state legislature.

Jimmie A. Gleason, M.D., Topeka, recognized Herman W. Hiesterman, M.D., Quinter, for his achievement as Alumni of the Year at UKSM. He also commended Steve Carter, Executive Director, for his excellent work during the two years he has held the position. The House expressed its concurrence in these commendations with a round of applause.

Dr. Stone noted two logistical problems which he believes interfere with the conduct of KMS Annual Meeting activities and prevent members from attending. He recommended that the following should be studied and preferably avoided in the future: scheduling of specialty society meetings in conflict with general activities; and scheduling the two sessions of the House of Delegates on consecutive days.

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The following Council District elections were announced:

District #1 — Wayne O. Wallace, Jr., M.D., Atchison, COUNCILOR

District #3 — James G. Bridgens, M.D., Shawnee Mission, COUNCILOR

District #5 — Frank C. Lyons, Jr., M.D., Manhattan, COUNCILOR; Rex R. Fischer, M.D., Manhattan, ALTERNATE

District #8 — Newton C. Smith, M.D., Arkansas City, COUNCILOR

District #9 — Jack E. Lungstrum, M.D., Salina, COUNCILOR

F. Calvin Bigler, M.D., Garden City, the newly installed KMS President, recognized the Speaker, Dr. Stone, and the Vice Speaker, Dr. Fransen, for the coming year. He listed the following activities as his priorities during his tenure:

- Membership recruitment
- Professional liability issue
- Development of young leadership by involving medical students and residents
- Interaction with governmental agencies
- Hospital relations
- Communications with and among physicians, with the public, media, other organizations, and government

- Active participation in the legislative process

He expressed his hope that organized medicine will take the initiative for leadership, and encouraged physicians to practice preventive as well as curative medicine. Dr. Bigler urged the members to read the *KMS Journal, Newsletter*, and other mailings in order to be fully informed of the many changes occurring in the field of medicine today, in areas of both science and economics.

Dr. Bigler announced that the appointment of committees has been completed and thanked those who have accepted the challenges of the coming year. He called on all physicians to unite and work in concert within the Kansas Medical Society for the benefit of the people of Kansas.

Dr. Stone expressed appreciation to members of the Reference Committee and to KMS staff for their work on the Annual Meeting.

Clair C. Conard, M.D., Dodge City, announced that the 1985 meeting will be in Colorado Springs. It is his hope that this will encourage more Kansas physicians to attend and become actively involved in the business of organized medicine.

The meeting was adjourned at 12:30 PM, to be followed by a meeting of the Council.

(Resolutions begin on page 201)

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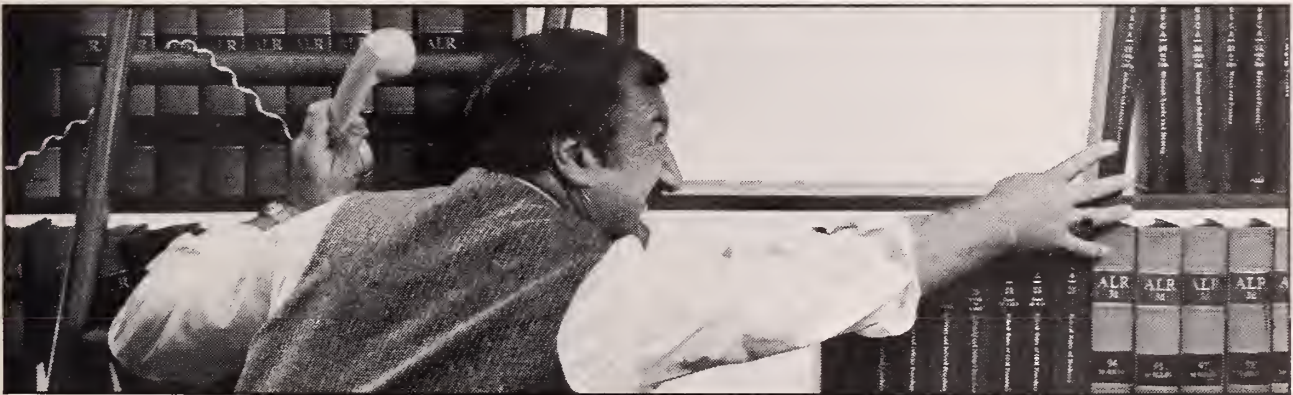
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Handicapped Children Referral in Kansas

MARY MIRA, Ph.D., *Kansas City, Kansas*, and KARL ALTMAN, Ph.D., *Lawrence*

CHILDREN who require early services are those who, through normal environmental interactions, may not be able to acquire the usual range of skills at a rate comparable to others of that age. Once identified, all handicapped children should be referred to rehabilitation services.¹ The specific types of disabilities include sensory and motor disabilities, behavioral abnormalities, mental retardation, learning disabilities, speech and language deficits, chronic health disorders, and children who are multi-handicapped. These children should be referred for rehabilitative services regardless of mildness or severity of the condition. The earlier the intervention the greater the benefit for the child and family.² Also included in this population are children who are at risk for problematic outcomes based on genetic, pre/peri-natal, or environmental factors.

During the 1982-83 school year, more than 42,000 children and youth below the age of 21 years with the above handicapping conditions were receiving special education and related services through the public schools in Kansas or in programs receiving funds through the Kansas Department of Education. A disproportionately small number of children below age 5 years were receiving services compared to those of public school age, 1,206 from ages 0-4 years inclusive vs 2,319 5-year-olds. This is a reflection of two factors. First, the laws do not mandate services for handicapped children below the age of 5 years in Kansas, and many providers may be unaware such services are available. Secondly, it is likely that there are a number of children ages 0-4 years not yet identified and/or referred to the service network. It is anticipated that more of these children will be identified and served as more and more primary care physicians are able to provide early and followup assessments and make knowledgeable referrals.

Listed here are the types of services generally indicated for handicapped children and resources to which the primary care physician may refer the child. An annual listing of these resources will be included in the KMS annual directory beginning in August 1984.

Screening and Identification Programs

The primary care physician usually manages the handicapped child's well-child care, acute care, and provides screening. However, the physician may wish to refer a child to a screening program for a variety of reasons: (1) if a disability is suspected because the child's development is not progressing normally; (2) if the family is high risk for a particular handicapping condition; or (3) if there is a question about the appropriateness of a particular rehabilitation service for a child. Screening programs function to recognize and treat conditions early enough to either reverse the disease process or slow its progression. Screening also identifies conditions other than the originally identified disease. The following are the major screening programs for children in Kansas.

Count Your Kid In

All local school districts or special education administrative units provide mandatory screening for *all* children suspected of being handicapped. This multidisciplinary screening is provided at least annually. Screening results and recommendations for further evaluations or placements are sent to appropriate agencies.

LOCATION: School districts or special education administrative units state wide

HOW TO REFER: Call local school district or 913-296-4951

Early Periodic Screening, Diagnosis, and Treatment (EPSDT)

Provides screening and referral for diagnostic and treatment services to children of families receiving Medicaid.

LOCATION: County health departments or designated providers

HOW TO REFER: Call local health department, SRS office, or EPSDT coordinator at 913-296-3981

Genetic Screening

Mandatory screening of newborns for PKU, hypothyroidism, and (as of January 1985) galactosemia, is provided in the newborn nursery. Screening for sickle cell anemia is available at the local level via the county health departments.

Address reprint requests to Dr. Mira, UKSM-KC, Children's Rehabilitation Unit, 39th & Rainbow Blvd., Kansas City KS 66103.

Comprehensive Evaluations

When a child has been identified as having a handicapping condition, the next step is a comprehensive assessment of the history of the problem and the child's current status. In addition to the comprehensive pediatric evaluation (*e.g.*, history and physical examination, developmental assessment) provided by the primary care physician, additional evaluations are frequently indicated and often required. Specifically, Public Law 94-142 requires a complete assessment of every child's sensory, motor, communication, and cognitive functioning prior to entering a special education program. This evaluation, with the information provided by the child's primary care physician, should not only define what special education program the child requires, but also should help to identify instructional goals and objectives. Public schools employ teams of specialists who can provide such evaluations. Even when an evaluation in a center other than the public school is indicated because of the uniqueness of the child's disability, the school is still responsible for the coordination of the evaluation. Resources for providing comprehensive interdisciplinary evaluations of handicapped children are described below.

Kansas Neurological Institute (KNI)

The Child Study Unit of KNI of Topeka provides short-term diagnostic services for individuals with mental retardation. Their multidisciplinary team includes an audiologist, nurse, occupational therapist, physical therapist, physician, psychologist, social worker, speech pathologist, and educational and vocational specialists. The resulting composite assessment of the child is communicated to the family and transmitted to appropriate agencies in the child's community to aid in the development of a treatment and training plan.

LOCATION: 3107 West 21st, Topeka, Kansas 66604

HOW TO REFER: Call 913-296-5377

University of Kansas Medical Center

The Children's Rehabilitation Unit, UKSM-Kansas City provides comprehensive in- or out-patient evaluation of handicapped children from birth to 21 years. The staff includes specialists in pediatric audiology, dentistry, neurology, nursing, nutrition, occupational and physical therapy, psychology, social work, special education, and speech pathology augmented by other medical specialists. Results of interdisciplinary evaluations are

made available to the primary care physician and local school personnel to assure a coordinated program of care for the child.

LOCATION: 39th and Rainbow Blvd., Kansas City, Kansas 66103

HOW TO REFER: Call 913-588-5926

Kansas Crippled and Chronically Ill Children's Program

The Crippled and Chronically Ill Children's Program in the Kansas Department of Health and Environment exists to promote the functional skills of Kansas youth under 21 years of age with physical handicaps, disabilities, or chronic diseases. The program provides or supports specialty health care for these youth and also for Kansas persons of any age who have chronic diseases of PKU, hemophilia, hypothyroidism, galactosemia (as of January, 1985), or sickle cell anemia. To promote quality of care, services are provided by professionals, hospitals, and vendors who meet identified standards of certification or licensure. All services of the program require prior authorization.

Diagnostic services are available to all Kansas youth from birth to 21 years of age who are suspected to have a handicap, disability, or chronic disease. Diagnostic services are provided through offices of private physicians, at specialty clinics of hospitals, and through outreach clinics across the State.

For additional information call the Crippled and Chronically Ill Children's Program, Department of Health and Environment — (913) 862-9360, Ex 455.

Regional Deaf-Blind Program

This program coordinates referral and maintains a listing of service providers for evaluating children suspected of dual hearing and vision deficits.

LOCATION: Regional throughout Kansas

HOW TO REFER: Call Penny Gottula, 913-296-2062 or 913-864-4570

Treatment Programs

Children with handicapping conditions require continuous, specialized services in rehabilitation and education. Services are available throughout Kansas to help the primary care physician provide assistance to children and families.

Kansas Crippled and Chronically Ill Children Treatment Service

Treatment services include medical specialty

care, hospitalization, laboratory, x-ray, durable medical equipment, and limited amounts of physical therapy, occupational therapy, and speech therapy for certain conditions. The medical conditions currently eligible for treatment include these: major orthopedic problems; burns; cleft palate/cleft lip; acquired or congenital heart disease; spina bifida; hearing loss; gastrointestinal or genito-urinary anomalies; and genetic diseases such as cystic fibrosis, sickle cell disease, PKU, hemophilia, hypothyroidism and galactosemia (as of January, 1985).

Case management service is offered to each child and family. This includes counseling, assistance in developing an individual plan of health care, and referral for other needed services. The program supports training for professionals who serve handicapped youth, alternative systems to deliver health care, and advocacy for the comprehensive needs, rights, and contributions of persons with handicaps, disabilities, or chronic diseases.

Application and information about services of the Crippled and Chronically Ill Children's Program are available at the administrative office in the Department of Health and Environment and at field offices located in Wichita, Kansas City, and Salina. Local health departments and social service departments of major hospitals can also facilitate referrals. The program has regulatory guidelines for financial and medical eligibility. Services are prior authorized within the context of an individual plan of health care that extends for a one year period.

HOW TO OBTAIN INFORMATION: Call (913) 862-9360, Ex 455

Head Start Preschools

A federal mandate stipulates that 10 per cent of the children served will be handicapped.

LOCATIONS: Throughout the state

HOW TO REFER: Call Ann Branden 1-800-332-0105

Public Schools

The major resource for rehabilitation and training for handicapped children is the public school. All children below age 21 years whom the primary care physician has identified as handicapped should be referred to the local special education district. For children below school age, many services are available which are funded through state special education funds and affiliated with local public schools. For children of school age, education is mandated for all handicapped children. In addition to educational programming — which may take place in

special classes, hospitals, home instruction, or regular classes with supplemental special services — public schools now also provide many ancillary rehabilitation services; *e.g.*, physical, occupational, and speech therapy. Additionally, many health care needs from conditions such as cystostomies and gastrostomies, which previously kept children out of school, are now often managed by school personnel.

When the primary care physician refers a handicapped child to the special education program of the local school, the physician can facilitate the referral by including the medical diagnosis, developmental history, description of the child's health status, and the implication of these for functioning in school and other environments. Once the child is placed in the special education district, the primary care physician's role in the total rehabilitative plan may include the development of a comprehensive health service plan, being available to advise the school on matters of the child's health, and insuring that the child's medical problems are being handled in the least restrictive manner.

LOCATIONS: Local school districts

HOW TO REFER: To identify local Director of Special Education, call KSDE 913-296-3866 or 800-332-6262

Private Residential Facilities

In addition to providing education and rehabilitative services on an inpatient basis, the following three facilities provide outpatient education and therapy for children in their catchment areas.

CAPPER FOUNDATION FOR CRIPPLED CHILDREN

LOCATION: 3500 West 10th, Topeka, KS 66604

HOW TO REFER: Call 913-272-4060

INSTITUTE OF LOGOPEDICS

LOCATION: 2400 Jardine, Wichita, KS 67219

HOW TO REFER: Call 316-262-8271, Ex 255

LAKEMARY CENTER

LOCATION: 100 Lakemary Drive, Box 449, Paola, KS 66071

HOW TO REFER: Call 913-294-4361

Family Support and Advocacy Programs

A handicapped child presents problems to the family which may not be resolved by meeting medical and educational needs. Parent programs benefit families by providing information, counseling, and assistance in securing services for children. Family support groups aid parents in addressing emotional issues related to a handicapped child, and allow opportunity for sharing concerns. When these parental needs are met, parents are more able to hear

and act on information from professionals about the child's disability.

Parent groups may be disability specific providing information, dissemination, counseling, lobbying, and advocacy. An example of such a program is:

THE ASSOCIATION FOR RETARDED CITIZENS, Kansas and local branches

LOCATION: Throughout the state

HOW TO REFER: Call Brent Glazier, 913-268-8200

Another type of support group meets the psychological needs of families. Examples are:

PARENT-TO-PARENT, a program pairing trained veteran parents with parents of newly diagnosed handicapped children to offer emotional support and information.

LOCATION: Kansas City

HOW TO REFER: Call the Association for Retarded Citizens and ask for Sonja Bowers, 913-648-2317

FAMILIES TOGETHER, INC. This program offers the total family of a handicapped child a chance to meet with other families for a weekend of combined educational and recreational activities.

LOCATION: Lawrence

HOW TO REFER: Call Chris Curry, 913-841-7241

FAMILY SERVICES: Trained providers offer respite

care either in the provider's home or in the home of a client for as few as three hours and up to 18 days per year.

LOCATION: Parsons University Affiliated Facility
HOW TO REFER: Call Marianne Adams, 800-362-0390

Accessing Resources for Children

In addition to the resource accesses already cited, there is a recently established Kansas SpecialNet Microcomputer System. SpecialNet is a nationwide communication network designed to provide current information for persons concerned with services and programs for handicapped individuals.

Kansas has a specific bulletin board called "KSHE" which features information of interest to local health professionals and educators. Information is available about: (1) specialists available for consultation; (2) resources for special disabilities and procedures; (3) specialized programs and outreach clinics; and (4) continuing education programs related to handicapped children and their families.

To access the Kansas SpecialNet system for information to help a specific child or to learn more about it, contact the local area special education director or call 800-332-6262.

References are available from Dr. Mira, UKSM-KC, Children's Rehabilitation Unit, 39th & Rainbow Blvd., Kansas City KS 66103.

Coming events in the JOURNAL . . .

- August issue — KMS Roster
- September issue — Papers from UKSM-Wichita

The President's Message

The Golden Glove Attack on the Doctrine of *Res Ipsa Loquitur*

The words *res ipsa loquitur* immediately attract the attention of the physician. The English translation of this Latin sentence is: "The thing speaks for itself." According to *Webster's Third New International Dictionary*, *res ipsa loquitur* is "a case in which mere proof that an accident took place is sufficient under the circumstances to warrant an inference that it was caused by defendant's negligence unless otherwise explained." This doctrine has been applied to several situations of medical liability where clearly an error has been made.

The Golden Glove Award is given annually to the best defensive baseball player at each fielding position in each major league. The manager, coaches, designated principal broadcaster, and principal sportswriter of each ball club vote for the league's outstanding fielding players each season. Frank White of the Kansas City Royals has won the Golden Glove Award several times, most recently in 1982. That year he had 361 put-outs, 389 assists, and 17 errors. The best defensive second baseman in the American League averaged one error for every 45 fielding chances.

What happens when a Golden Glove winner, or any major league baseball player, makes an error? Does one mistake mean negligence? Does the ballplayer get sued? He does get credit for making as few errors as possible.

The two best defensive baseball teams in all major league history were the Baltimore Orioles of 1964 and 1980. Each year the team made only 95 errors in more than 6,300 fielding chances, for an average of three errors per 200 chances. Yet the 1980 Orioles did not win the World Series. They did not even win the pennant. They were second in the Eastern Divi-



sion of the American League. The 1964 team finished third. Something more was needed to be a winner than playing relatively error-free ball.

The public is becoming more realistic and is aware that physicians can err. Perhaps we physicians would have more crowd appeal — as well as be more human and humane — if we dropped the aura of infallibility. Should there be an annual award in medicine for the fewest errors possible in each team position?

F. Calvin Buzby, M.D.
President



Fragile Franchise

The computer rapped out its dotted information in an AMA newsletter that the AMA and AMA Auxiliary would be pushing a vigorous voter registration drive (vigorous drive, that is, although one would hope that the voters would be, too). Now being the political season, this is not a surprising message. What followed *was* surprising — that in “some areas of the country, as many as 40 per cent of eligible physicians have not registered to vote.”

The authority for this statement is not cited but one dismisses it only at the risk of entertaining the less supportable belief that physician involvement in the electoral process is as nearly total as it should be. The relatively low response of eligible voters in most elections in this country has long inspired reactions ranging from the specious defense that withholding the vote is in itself an exercise of the democratic spirit to exasperated demands that voting be forcefully required, itself inherently destructive to that spirit. Those who contend that their failure to vote is a gesture against the times, the issues, the persons, or whatever, fail to understand the essence of democracy. It is not a palpable, free-standing entity to be used when expedient and put on “hold” otherwise. Only as it is used positively is it substantive, and failure to exercise it automatically results in its replacement by some less desirable system. It is usually cumbersome, often imprecise and, to totalitarians, inefficient as compared with the forced certainties of their systems.

The failure of many people to exercise this important franchise has long puzzled students of politics. Perhaps it is a civic softness from generations of easy privilege. Certainly, it is valued most by those who are denied it or have to fight for it. But even other democracies are bemused by the American system. The American public, subjected to overlong, overloud, and overpriced political campaigns might be expected to seek to have the multiple choice statement, “None of the above,” added to the ballot but

this is not a viable option in the democratic sense.

We suspect that of greater effect is a sense of futility born of a seeming detachment of the political process of voting from its ultimate translation into practical effect on our lives — not just the visible performance of public officials or the publicized statutory products of the system but also the countless ways in which the bureaucratic disseminations of the effort ultimately move our lives in the social currents. Of necessity, elected officials now rely greatly on staffs for research, development of legislation, and confronting the public. Accomplished legislation is administered by the bureaucratic monster. Thus, the benefits and intentions envisaged in the electoral process are, after all, largely effected by non-elected individuals.

Turning to medical practice, one does not think ordinarily of the voting process as being an integral part of the physician-patient relationship. Yet, all are aware that there is no phase of the vital physician service that is not subject to the current political tides — the methods of delivery of medical care, the quantity and quality of medical training, the remuneration, both method and substance, control of material and personnel. How can it be otherwise?

But democracy does not guarantee all things to all people (although the idea has contributed more than a little to our present problems). Cumbersome though it is, it continues to be the best mechanism over the long run for converting public wishes to public action even if at times it requires the rescinding of previous public action or proves that the public voice is not always right. More important than any election or the issues or the individuals involved is the fact that democracy, to fulfill its purpose and promise, is a full-time job. The same can be said for the physician-patient relationship.

It suggests a paraphrase of the old maxim about reading: The person who does not vote has no advantage over the person who cannot vote. — *D.E.G.*

Joint Position Statement on Long Term Care

*Kansas Medical Society
Kansas Department of Health & Environment
Kansas Department on Aging
Kansas Department of Social & Rehabilitation Services*

PROJECTED INCREASES in the number and proportion of the elderly will be one of the most critical factors affecting the health care system during the next several decades. An understanding of the key issues and developments in the delivery and financing of care for the aged will help all concerned respond to a changing environment. This joint position statement provides a broad assessment of issues in long-term care.

Issues in Long Term Care

Long term care refers to any professional or personal service required on a recurring or continuous basis by an individual because of chronic or permanent physical and/or mental impairments. Three components are implicit in the definition: (1) the provision of a variety of medical, health, social, and general support services; (2) the involvement of families, friends, professionals, paraprofessionals, and volunteer service providers; and (3) service delivery in such settings as the home, community agencies, specialized sites, and institutions. The array of services, providers, and settings involved in long term care has led to use of the phrase "continuum of care" to describe the system.

During the last decade, the provision of long term care services emerged as one of the most important health and social concerns. This is due to a number of factors. In brief:

- Substantial increases have taken place in the size and proportion of the elderly population, defined as persons age 65 years and older. In 1900, 4 per cent of the population (3 million persons) was elderly. Currently, 11.3 per cent of the United States population (25.5 million persons) is elderly; in Kansas, more than 13 per cent of the population (approximately 306,300 persons) is age 65 years or older. During the early 21st century, the proportion of elderly will be 18-22 per cent of the total population; further, close to half of the elderly will be in high-risk categories due to advanced age (75 years or older).
- Utilization of health services increases with age. Four out of five older persons have one or more chronic ill health conditions; older persons are

also affected by acute health problems (especially respiratory infections) and accidental injuries (especially falls leading to fractures of the hip). The elderly see physicians twice as often as young people (6.3 visits per year vs 3.2); they are hospitalized more frequently (18 per cent per year vs 11 per cent for persons under 65); and they spend more than \$2,600 per year on health care, nearly 7.5 times more than young people.

Until very recently, long term care was considered to be health-related services provided in various types of institutions, primarily nursing homes. In fact, only 5 per cent of the United States elderly and approximately 7 per cent of Kansas elderly reside in nursing homes. Further, between 10 and 40 per cent of the elderly residing in nursing homes are estimated to be capable of returning to the community if appropriate support services were available.

Gradually, long term care has been expanded to include services to chronically ill and impaired persons to enable them to live outside institutions. Some of the services include: home health, adult day care, homemaker care, transportation services, meal programs, and alternative forms of housing (foster care, respite care, etc.). The services thus involved — particularly the less medically-oriented personal care and supportive services — can be provided either formally by individuals or agencies who are paid for their services, or informally by relatives or friends without pay. In regard to the latter, studies by the General Accounting Office indicate that 60 to 80 per cent of long term care is informally provided by spouses, other relatives, or friends.

Failure to consider the full range of long term care services, providers, and settings will result, as previously noted, in the unnecessary institutionalization of elderly persons who could otherwise receive needed medical care while living at home or in the community. Unnecessary nursing home placement also creates problems for patients who remain in a hospital because a needed nursing home bed is unavailable. Implementation of a diagnostic-related grouping (DRG) payment system by Medicare may exacerbate this concern as physicians and hospitals face new pressure to treat and release patients quick-

ly. It must also be noted that broader coverage of in-home and community-based social services may be expensive. There is little evidence that coverage of a range of services will substantially reduce *total* health care expenditures. This is because of increases taking place in the size of the elderly population, and to a degree, expanded service benefits have resulted in new, additional service populations in need rather than one-to-one substitutions for nursing home care.

Society approaches the 21st century at a time when increasing scientific attention is being given to the interplay among social conditions, biological factors, and psychological issues of aging. It is becoming increasingly clear that aging is, in fact, a biopsychosocial life process; *i.e.*, there is no single cause of the problems of aging. The ability to make available a broad range of social support and health services to assure care and treatment in the least restrictive, most appropriate setting will be a major issue of the future. The demographic changes discussed, coupled with differences in longevity between men and women, an increasing divorce rate, a declining birth rate, and increasing participation of women in the work force, all point to stresses that will affect the long term care system.

Policy Position Proposal

In response to the emerging socioeconomic trends affecting the delivery of medical and health-related services to the elderly and others, the following policy position proposal is set forth.

Long Term Care System Goal: To ensure that individuals in need of long term care have access to a continuum of informal and formal services that will promote physical, social, and psychological functioning by helping the individual to cope with disabilities and live as independently and normally as possible.

Objective No. 1

A continuum of long term care service should exist in Kansas communities so that there are alternatives to institutional care.

Proposed Activity — Specific Service Needs

The potential cost of providing a continuum of long term care services must be balanced against quality considerations: the quality of care the population deserves and the quality of life for people in need of long term care services. The following actions are proposed:

- Kansas communities should have a core set of

long term care services available. The minimum service set should include: home health, home-maker services, income programs, meal programs, day care, transportation services, nursing homes, hospitals, alternative housing, and case management/service coordination.

- The Kansas Legislature should continue to provide seed money for the development of long term care core services.
- Kansas communities should take the initiative to develop plans for ensuring that a continuum of formal and informal services is available to the elderly by establishing task forces involving the elderly and community service providers. One example of how this is currently being done is the Kansas Department of Health and Environment demonstration program, LIVELY.

Proposed Activity — Case Management

The current long term care system consists of a confusing and fragmented array of services. A coordinating mechanism/case management service is needed to assist individual and families in assessing needed services and in identifying the potential for problem situations. The purpose of the service is to help the individual maximize abilities, not to take over all decisions. The functional components of case management include:

- Client identification;
- Multidisciplinary assessment of client service needs;
- Coordinated care planning for the client;
- Care plan implementation; and
- Care plan monitoring/followup.

Proposed Activity — Reimbursement System Changes

Development of a full continuum of long term care services will be of little value if reimbursement systems do not support the services. Activities should be undertaken in three areas:

- Private sector approaches to financing and delivering services for the aged should be encouraged.
- Public sector (Medicare and Medicaid) financing should emphasize the full range of health and social services needed by the elderly. The Kansas Medicaid Home and Community-Based Services (HCBS) waiver program is one example of how change can take place.
- Public and private sector demonstration programs should emphasize alternative service delivery and financing. Social/health maintenance organiza-

tion projects which provide care for a prepaid amount are an example. Consideration of compensation (in the form of tax incentives or deductions) for home care provided by families is another example.

Objective No. 2

Education programs for health professionals should contain mandated, structured content on geriatric care.

Proposed Activity — Medical Education

The University of Kansas Medical School should establish clear objectives in the teaching of geriatric medicine.

Proposed Activity — Continuing Education

Continuing education should stress alternative medical technologies and delivery mechanisms aimed at reducing unnecessary costs associated with the care of the very old and terminally ill; *i.e.*, the difference between care and cure philosophies.

Organized medical education is needed for the health profession and the public on the chronic, multiple afflictions affecting some elderly, and the socioeconomic implications involved in caring for this population. Private foundation funding sources should be explored for this purpose.

Objective No. 3

A comprehensive, coordinated state policy on long term care must be developed and actively promoted by a partnership of the public and private health sectors.

Proposed Activity — Policy Committee

Several years ago the Governor established a cabinet subcommittee on long term care comprised of the secretaries of Health and Environment, Aging, Social and Rehabilitation Services and Transportation, as well as the Director of the Budget and the Chancellor of the University of Kansas. Concerns have been expressed that this policy body does not have specific provisions for input by private professional organizations involved in long term care service delivery. Options for providing such input range from establishment of a special coalition of private and professional group/associations concerned with aging issues, which would develop and share policy concerns with the cabinet subcommittee, to establishing a body of major organizations and government agencies that impact on long term care.

Summary

Actions are needed in three areas to ensure that Kansas elderly have access to a continuum of informal and formal long term care services. First, in-home and community-based services such as home health, homemaker care, meal programs, day care, and transportation are needed in many communities as possible alternatives to institutional care. To achieve this aim, Kansas communities should establish task forces to assess their present system and develop future plans, and the Kansas Legislature should assist by continuing to provide some seed money for new service development. Case management/service coordination programs are needed to help overcome problems of system fragmentation, and public and private reimbursement sources should encourage the provision of the entire continuum of care.

Second, education programs for both health professionals and the general public are needed so that there is greater awareness of geriatric issues. The University of Kansas Medical School should establish clear geriatric teaching objectives, and continuing education is needed on alternative technologies and delivery mechanisms for long term care. Finally, a comprehensive, coordinated state policy on long term care should be developed and actively promoted by the public and private health sectors.

Resolutions

An asterisk following the resolution number indicates a change in the Constitution and By-Laws.

RESOLUTION NO. 84-1*

Sunset of 1979 Resolutions

KMS By-Laws — 5.444

“Official policies established through resolutions at the House of Delegates shall be in effect for a period of five (5) years, at which time that policy position will be reviewed by the Executive Committee and will expire subject to the approval by the House of Delegates unless superseded or continued by another resolution.”

Resolved, That the following 1979 Resolutions be readopted:

1. General Fund Check Authorization
4. AMA Dues Billing and Remittance Criteria
19. Seat Belt Restraints for Infants and Children (see Resolution 84-9)

20. Probationary Members
21. Associate Membership Category
22. Component and State Society Membership
24. Judicial Committee (see Resolution 84-15);

and be it further

Resolved, That the following 1979 Resolutions expire:

2. AAMA 1980 Meeting
3. Medical Ethics and Chiropractic
5. Medical Care Costs
6. Hospital Rate Review
10. Chiropractic Venipuncture Privilege
11. Second Opinion Surgical Programs
13. Federal Regionalism
15. Committee to Study CME
16. Current CME System
17. Breast Feeding
18. International Year of the Child
23. Relations with Specialty Societies
25. James E. Hill
26. CME for Nurses
27. Nursing Education
29. Kansas Medicaid Program
30. Health Care Cost Containment
31. Professional Liability Cases
32. Reno County Medical Society and Auxiliary

RESOLUTION NO. 84-2

Control of Pediculosis

WHEREAS, The current "Kansas Statutes and Regulations Regarding Communicable Disease Control" requires that "students infested with lice shall be excluded from school or child care facilities until treated with an antiparasitic drug, and until all nits have been removed"; and

WHEREAS, This required exclusion from school can involve as much as two weeks; and

WHEREAS, The preponderance of medical literature recommends that the students' exclusion from school is not necessary for control purposes and recommends that students having pediculosis should be readmitted to school after the first treatment; therefore be it

Resolved, That the Kansas Medical Society endorse the position that students having pediculosis be allowed to return to school the morning after the student has been treated with an antiparasitic medication; and be it further

Resolved, That the Kansas Medical Society initiate appropriate action to effect deletion from the

"Kansas Statutes and Regulations Regarding Communicable Disease Control" of the requirement that all nits must be removed before the student may return to school.

RESOLUTION NO. 84-3

Disaster Preparedness

Not adopted.

RESOLUTION NO. 84-4

Support of the Civilian Military Contingency Hospital System (CMCHS)

WHEREAS, The United States government once maintained one of the largest military medical systems in the world (more than 400,000 beds in 1945) and for economic reasons now maintains one of the smallest systems (16,000 beds in the U. S. and 2,000 abroad) which is barely enough to meet peacetime needs of healthy, unassaulted military personnel and dependents; and

WHEREAS, This huge deficit of beds in wartime will be largely met by using the current surplus of civilian hospital beds through the Pentagon's CIVILIAN-MILITARY CONTINGENCY HOSPITAL SYSTEM (CMCHS, pronounced "sim-chis") of 56,000 beds, similar to the excellent civilian-military system developed in Israel; and

WHEREAS, In the time of war, CMCHS will not be just an *adjunct* to the military hospital system, it may largely *become* the military system which will have to be implemented easily and efficiently to handle large numbers of severely wounded casualties with illnesses and injuries not ordinarily seen in civilian life; and

WHEREAS, This system was initiated more than three years ago following approval by both American Medical Association and American Hospital Association in 1980, but still remains largely unknown to most physicians, hospitals, and even many military personnel despite its crucial and widely acknowledged importance to our sons and daughters and even the potential survival of our society; therefore be it

Resolved, That the Kansas Medical Society recommend to the Kansas Hospital Association and the Kansas Military Authorities that the Civilian Military Hospital System be clarified; and be it further

Resolved, That the Kansas delegation to the AMA be instructed to present a similar national resolution

for ongoing Pentagon liaison and support of CMCHS nationwide; and be it further

Resolved, That the Kansas Medical Society make its membership aware of CMCHS and incorporate it into local medical society disaster plans.

RESOLUTION NO. 84-5

Preparticipation Health Evaluation for School Athletics

WHEREAS, Participation in Kansas school athletics has become increasingly popular among both boys and girls; and

WHEREAS, Competition in sports is needed and enjoyed by young people for status, personal satisfaction, and social acceptance; and

WHEREAS, Proper preparticipation medical evaluation of children and youth who engage in athletics is essential to prevent illness or injury; and

WHEREAS, An advisory committee of physicians, nurses, coaches, trainers, and other professionals has developed updated recommendations regarding preparticipation athletic evaluation to replace the form presently recommended by KSHAA; and

WHEREAS, These new forms are specifically designed to protect the student athlete from illness or injury; therefore be it

Resolved, That the Kansas Medical Society endorse a preparticipation sports evaluation and examination form to prevent injury and illness; and be it further

Resolved, That the Kansas Medical Society work with the Kansas State High School Activities Association to develop an appropriate examination form.

RESOLUTION NO. 84-6

KMS Commendation

WHEREAS, The House of Delegates voted to establish a Medical Student Section of the Kansas Medical Society at the 1983 Annual Meeting; and

WHEREAS, This resolution established provision for one delegate and one alternate delegate to the House of Delegates of the Kansas Medical Society; and

WHEREAS, This action offers the opportunity for future physicians to participate actively in organized medicine and demonstrates interested concern by practicing physicians for the future of the Medical Profession in the State of Kansas; therefore be it

Resolved, That the Kansas Medical Society

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accept commendation and thanks from the Medical Student Section members for encouraging active participation by members of the profession through the formal establishment of a Medical Student Section and positions of delegate and alternate delegate to the House.

RESOLUTION NO. 84-7

Quality of Medical Education

WHEREAS, The quality of medical education can be severely compromised by less individualized attention in the first two years of basic sciences and by less clinical exposure during the final two years when a large student population is competing for exposure to a limited patient population; and

WHEREAS, Statistics demonstrate that the state in which a residency position is held is a better predictor for permanent location of a physician's future practice than is location of undergraduate medical education; therefore be it

Resolved, That the Kansas Medical Society appoint a committee to work in cooperation with the Kansas Board of Regents and the University of Kansas to study the quality of medical education at the University of Kansas and evaluate the implications of entering medical class size; and be it further

Resolved, That a position paper considering these topics be prepared and made available to the Executive Vice Chancellor of the College of Health Sciences at the University of Kansas and to the Board of Regents for the State of Kansas.

RESOLUTION NO. 84-8

University of Kansas Medical Center

WHEREAS, The University of Kansas Medical Center was established primarily for the training of physicians and related professionals necessary to meet the medical needs of the State of Kansas; and

WHEREAS, The expansion of facilities has now exceeded these needs and presents a significant financial burden for both the facility and the State; and

WHEREAS, The institution should serve as the hub of medical education and research activity for the community and the state; and

WHEREAS, There needs to be better communication and cooperation between the medical community and the Medical Center so as to develop an

expanded area of support; therefore be it

Resolved, That the Kansas Medical Society continue its liaison committee of interested, informed physicians who will investigate and make recommendations relative to the current training needs of the state as related to the current activities of the Medical Center.

RESOLUTION NO. 84-9

Child Passenger Safety Restraints

Withdrawn by sponsor.

RESOLUTION NO. 84-10

Smoking Prohibition During KMS Meetings

WHEREAS, Physicians are in a unique position to serve as role models for the public by practicing good health habits; and

WHEREAS, The Kansas Medical Society historically supports issues that enhance health care and quality of life for all citizens of the State of Kansas; and

WHEREAS, Cigarette smoking has been incontrovertibly linked to a wide variety of severe and life-threatening health consequences; and

WHEREAS, Resolution 121, A-83 of the AMA House of Delegates encourages a smoke-free society by the year 2000 and several resolutions are currently being considered by the AMA Board of Trustees to speed this process; therefore be it

Resolved, That the smoking of cigarettes be prohibited at all formal meetings of the Kansas Medical Society and standing committees of the Society by the year 1990; and be it further

Resolved, That in the interim, all members attending meetings of the Society be encouraged to refrain from smoking during the meetings.

RESOLUTION NO. 84-11

Student Representation on KMS Committees

WHEREAS, During this first year of functioning of the Medical Student Section (MSS), students have demonstrated enthusiastic interest in participating in organized medicine on the many levels made available through the Section; and

WHEREAS, Participation in organized medicine offers unique opportunities for broadening profes-

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sional education and promoting lifelong involvement by teaching students the responsibilities of political awareness; and

WHEREAS, Many of the business functions of the Society are conducted by committee; therefore be it

Resolved, That provision be made by the Society for the establishment of committee positions as voting members for students as deemed appropriate by the Executive Committee to enhance communication and involvement of the student members in the functioning of the Society; and be it further

Resolved, That the Governing Council of the MSS shall establish the guidelines for the selection of student representatives to these committees to be submitted to the MSS members for incorporation into the MSS by-laws.

RESOLUTION NO. 84-12A

Medical Student and Resident Physician Sections

WHEREAS, The Kansas Medical Society in 1983 established student and resident sections; and

WHEREAS, It has been shown that early involvement in organized medicine is the best guarantee of sustained commitment to organized medicine; therefore be it

Resolved, That the Membership Committee work to activate the Medical Student and Resident Physician Sections in 1984-85; and be it further

Resolved, That a budget be allocated for expenses; and be it further

Resolved, That the KMS appoint students and residents to serve as voting members on appropriate committees; and be it further

Resolved, That component medical societies encourage student and resident involvement on the local level.

RESOLUTION NO. 84-12B

Medical Student Dues

No adopted.

RESOLUTION NO. 84-13*

Washington County

Resolved, That the By-Laws be amended as follows:

Add Washington County to Council District #1.

Delete Washington County from Council District #5.

RESOLUTION NO. 84-14*

Hospital Medical Staff Section

Resolved, That the following be added to the By-Laws: 4.5822 Hospital Medical Staff Section

Purpose. The purpose of this section is to provide a direct means to address the relationship between members of the Kansas Medical Society and hospital staffs.

Membership. Membership in the section shall be limited to KMS members selected by physician members of the medical staffs of hospitals.

Governing Council. There shall be a Governing Council of the Hospital Medical Staff Section to direct the programs and activities of the section, subject to the approval of the KMS Council.

Members. There shall be five voting members of the Governing Council, consisting of the officers, delegate and alternate delegate, elected at the business meeting of the section.

Officers. The officers of the section shall have the following duties and responsibilities.

Chairman. The Chairman shall preside at the business meetings of the section and at meetings of the Governing Council.

Vice Chairman. The Vice Chairman shall assist the Chairman and preside in the absence of the Chairman or at his/her request.

Secretary-Treasurer. The Secretary-Treasurer shall maintain such records and accounts as may be necessary or advisable for the conduct of the activities of the section.

Delegate and Alternate Delegate. The Delegate and Alternate Delegate shall represent the members of the section in the KMS House of Delegates.

Term. Governing Council members, including the Delegate and Alternate Delegate, shall serve staggered three-year terms, beginning at the conclusion of the Annual Meeting at which they were elected.

Vacancies. Any vacancy occurring on the Governing Council shall be filled at the next business meeting of the Section.

Business Meeting. There shall be a business meeting of members of the section held prior to each Annual Meeting of the House of Delegates.

Representatives to the Business Meeting. The physician members of the medical staff of each hospital may select a representative to the business

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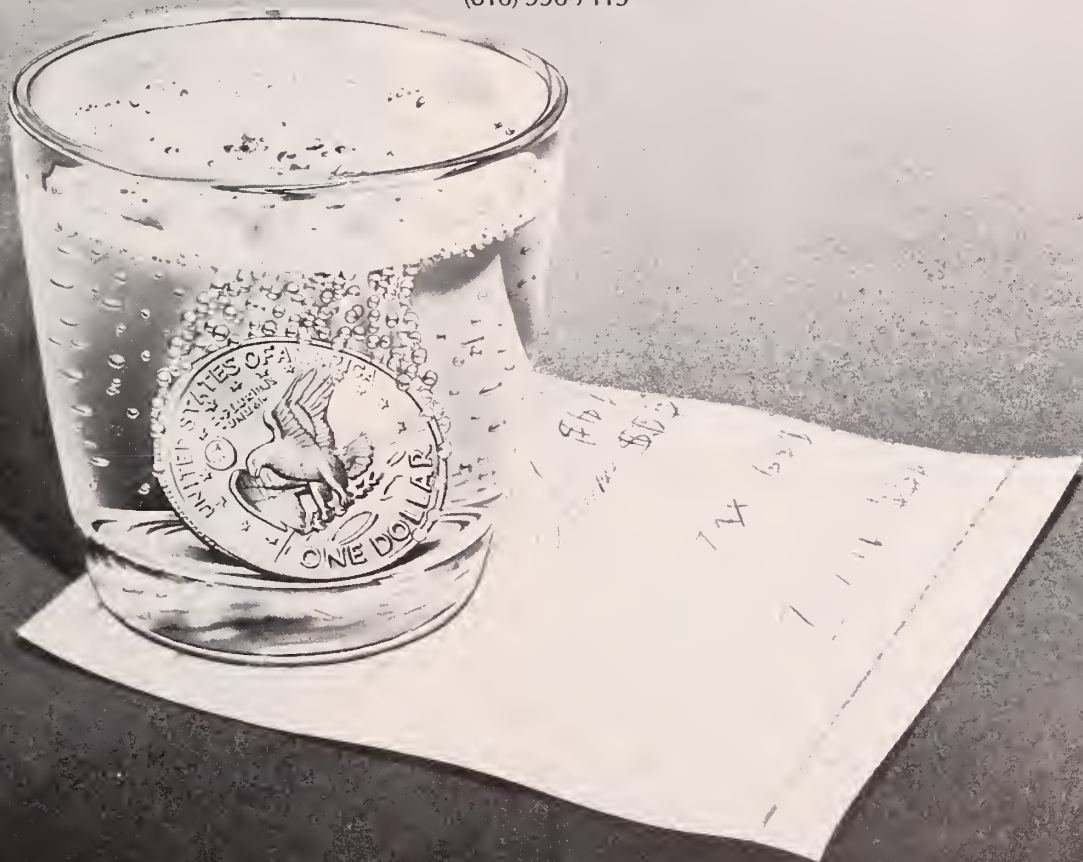
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meeting of the Hospital Medical Staff Section. The representative must be a KMS member who is an active voting member of the medical staff with clinical privileges at the hospital.

Representatives to the business meeting shall be elected by and from the active voting members of the medical staff of each hospital.

Representatives to the business meeting shall be properly certified by the President or Secretary of the medical staff.

Only duly selected representatives to the Hospital Medical Staff Section shall have the right to vote at the business meeting of the section, but the meeting shall be open to any member of the Kansas Medical Society. The meeting shall be conducted pursuant to rules of procedure adopted by the Governing Council of the section and approved by the KMS Council.

Purposes of the Business Meeting. The purposes of the business meeting shall be:

- a. To hear such reports as may be appropriate.
- b. To consider and vote upon such matters as may properly come before the meeting.
- c. To adopt resolutions for submission by the section to the House of Delegates of the Kansas Medical Society.
- d. To elect at the business meeting prior to the Annual Meeting of the KMS House of Delegates a Chairman, Vice Chairman, Secretary-Treasurer, Delegate and Alternate Delegate.
- e. To conduct such other business as may properly come before the meeting.

RESOLUTION NO. 84-15*

Judicial Committee

Resolved, That the By-Laws be amended to read as follows:

9.1 Judicial Committee

There shall be a Judicial Committee, representing different specialties. One member of the committee shall be a physician representing the Kansas State Board of Healing Arts. The President shall appoint the committee subject to approval by the Council. The majority of the Judicial Committee shall constitute a quorum and the affirmative vote of a majority of those members present shall constitute the action of the Judicial Committee.

- 9.6 Upon reaching a decision, the Judicial Committee chairman shall transmit to the Council and

the Board of Healing Arts a written statement and explanation of the final decision as soon as possible after the committee has completed the investigation of the case and has arrived at its decision.

RESOLUTION NO. 84-16*

Editorial Board — By-Laws Change

Resolved, That the By-Laws be amended to read as follows:

10.0 The Editorial Board

- 10.1 The Editorial Board is comprised of members of the Society appointed by the Council. When a vacancy occurs, it is to be filled, if necessary, at the next meeting of the Council.

10.2 Duties of the Chairman

- 10.21 To direct the operation of the Board and be Editor of *The Journal of the Kansas Medical Society*.
- 10.22 To make reports to the House and Council of the activities of the Board.

10.3 Duties of the Board

- 10.31 To perform the functions necessary to the publication of the *Journal* and other publications as considered necessary by the House of Delegates or the Council.
- 10.32 The Board may appoint associate editors from any Council district for particular functions in fulfillment of its duties.
- 10.33 The Board with its associate editors shall meet at the call of the Chairman.
- 10.34 The Board will serve as the Necrology Committee as directed by the House of Delegates or Council.

10.4 Financial Regulations

- 10.41 The Board is enjoined to maintain the *Journal* within budgetary limits established by the Council.
- 10.42 Expenditures authorized by the Board are to be paid by the Kansas Medical Society upon authorization of the Business Manager of the *Journal*.



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RESOLUTION NO. 84-17**KMS Journal Budget**

Not adopted.

RESOLUTION NO. 84-18**Medicare Funding of Diagnosis Related Groups**

WHEREAS, The Federal Medicare program through its use of Diagnosis Related Groups (DRGs) discriminates between rural and urban hospitals using regional wage indexes and baseline urban and rural pricing which economically disfavor rural hospitals greatly; and

WHEREAS, This funding practice may result in the economic failure of some hospitals; and

WHEREAS, Health care shortages and medically underserved areas of this country occur predominately in rural localities; and

WHEREAS, The taxation funding this program is shared equally by all taxpaying citizens, regardless of whether they live in a rural or urban area and regardless of any local wage index; therefore be it

Resolved, That the Kansas Medical Society oppose the present Diagnostic Related Group funding of Medicare patients to hospitals based on geography alone and support a uniform and equitable payment method; and be it further

Resolved, That this resolution be forwarded to the AMA for its action at the annual 1984 House of Delegates Meeting.

RESOLUTION NO. 84-19**Hospital Outpatient Clinics**

WHEREAS, Hospitals across the country are increasingly becoming involved in the private practice of medicine at an accelerating rate; and

WHEREAS, Satellite and commercial medical mall clinics sponsored by both hospitals and physicians are increasing at a rapid rate; and

WHEREAS, Federal law prohibits any restraint or action that might discourage market entry and deter experimentation and new developments by individual entrepreneurs; and

WHEREAS, The AMA Committee on Medicolegal Problems will complete a three-year study of professional and legal problems posed by the emergence of commercial forms of medical practice and report its findings at the AMA Annual Meeting, June, 1984; therefore be it

Resolved, That the Kansas Medical Society distribute the 1984 final report of the AMA Committee on Medicolegal Problems to county medical societies, KMS committees and KMS Councilors for review and comments; and be it further

Resolved, That the AMA Delegates of the Kansas Medical Society review the report of the AMA Committee on Medicolegal Problems, review comments of KMS members, and make recommendations to the KMS Executive Committee and Council regarding this report.

RESOLUTION NO. 84-20**In-Hospital Required Forms**

Not adopted.

RESOLUTION NO. 84-21**Kansas Foundation for Medical Care**

WHEREAS, Some physicians have voiced concern regarding the potential loss of input by the Kansas Medical Society in the overall operation of the Kansas Foundation for Medical Care; and

WHEREAS, Physicians support cost containment efforts as long as patient care is not jeopardized; and

WHEREAS, History has proven that with continued and active physician input, medical programs are more likely to achieve their intended goals; therefore be it

Resolved, That the Council of the Kansas Medical Society develop recommendations identifying specific areas or guidelines for increased participation of the Kansas Medical Society in the overall management and operation of the Kansas Foundation for Medical Care so as to assure that the operation of the Kansas Foundation for Medical Care represents the philosophy and views of Kansas physicians and that patient care remains the top priority when implementing all Foundation review programs.

RESOLUTION NO. 84-22**Chiropractic**

(Not adopted — Referred for study)

WHEREAS, The interpretation of the scope of chiropractic practice is vague and misleading to the public, employers, third party payers, licensing agencies, and government; and

WHEREAS, It appears that this confusion should be eliminated, therefore be it

Resolved, That an interim study be conducted by a committee of the Kansas Legislature for the purpose of defining the scope of chiropractic practice. This study should include a detailed examination of what is taught in chiropractic colleges to determine what chiropractors can legitimately do.

RESOLUTION NO. 84-23

Alternative Medical Organization

WHEREAS, The long-standing goal of the medical profession has been to provide quality care to the sick and injured based on free choice of physician and hospital at reasonable costs; and

WHEREAS, The environment of medicine is changing through implementation of HMOs, PPOs, DRGs, and the like; and

WHEREAS, It appears that the effects of these new health care delivery and cost containment programs could create an atmosphere that would adversely affect the time honored goals of the profession; therefore be it

Resolved, That the KMS Council fully evaluate the pros and cons of establishing an alternative organization for the purpose of safeguarding the goals of the private practice of medicine and overall patient care and that this report be submitted to the KMS House of Delegates at their 1985 meeting.

RESOLUTION NO. 84-24

Professional Liability

Resolved, That the following proposals be adopted:

- Study the private insurance market to determine if there are viable alternatives to continuing the Health Care Stabilization Fund.
- Study the issue of the Health Care Stabilization Fund providing tail coverage for resident physicians leaving the state and retiring physicians with less than a certain minimum time of participation in the HCSF.
- Support the following tort reform proposals in the next legislative session:
 - Limit on total awards
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 - Abolishment of the collateral source rule
 - Limitation of attorneys' fees

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- Reduction of the current judgment interest rate of 15 per cent
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- Expiration of scheduled periodic payments on death of the plaintiff

RESOLUTION NO. 84-25

Local Health Departments

WHEREAS, According to Kansas Statutes 65-201, "The county commissioners . . . shall act as county boards of health for their respective counties. . . . Each county board thus created shall appoint a person . . . who shall serve in an advisory capacity to the county board of health and as the local health officer. . . . The local health officer . . . shall hold office at the pleasure of the board"; and

WHEREAS, The funds provided to the local county health departments by Kansas Department of Health and Environment are based on and in accordance with the locally developed plan; and

WHEREAS, The local health care plan is the responsibility of the local health commission and/or their designee; and

WHEREAS, The health of the public is logically the mutual concern of the private practitioners and the public health departments; and

WHEREAS, Communication between these two sectors varies throughout the state from very good to very poor and could be improved by such suggestions as:

- Inviting local health departments to make annual or more frequent presentations to local medical societies about their services and problems;
- Encouraging physicians to respond to requests from local health departments for assistance and advice;
- Encouraging physicians to serve willingly and conscientiously as health officers when asked;
- Encouraging cooperation between local health departments and local physicians in determining the scope and standards of care provided under their auspices;
- Encouraging local physicians to develop better methods of reporting communicable diseases, environmental diseases, and environmental and other community health problems; and
- Encouraging physicians to help educate local health personnel as to local standards of health care; therefore be it

References:

1. Stone PH, Turri ZG, Muller JE. Efficacy of nifedipine therapy for refractory angina pectoris. *Am Heart J* 104 672-681. September 1982
2. Antman E, Muller J, Goldberg S, et al. Nifedipine therapy for coronary artery spasm. Experience in 127 patients. *N Engl J Med* 302 1269-1273. June 5, 1980

BRIEF SUMMARY

PROCARDIA® (nifedipine) CAPSULES

For Oral Use

INDICATIONS AND USAGE: 1. **Vasospastic Angina:** PROCARDIA (nifedipine) is indicated for the management of vasospastic angina confirmed by any of the following criteria: 1) classical pattern of angina at rest accompanied by ST segment elevation; 2) angina or coronary artery spasm provoked by ergonovine; or 3) angiographically demonstrated coronary artery spasm. In those patients who have had angiography, the presence of significant fixed obstructive disease is not incompatible with the diagnosis of vasospastic angina, provided that the above criteria are satisfied. PROCARDIA may also be used where the clinical presentation suggests a possible vasospastic component but where vasospasm has not been confirmed, e.g., where pain has a variable threshold on exertion or in unstable angina where electrocardiographic findings are compatible with intermittent vasospasm or when angina is refractory to nitrates and/or adequate doses of beta blockers.

2. **Chronic Stable Angina (Classical Effort-Associated Angina):** PROCARDIA is indicated for the management of chronic stable angina (effort-associated angina) without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or organic nitrates or who cannot tolerate those agents.

In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in those patients are incomplete.

Controlled studies in small numbers of patients suggest concomitant use of PROCARDIA and beta blocking agents may be beneficial in patients with chronic stable angina, but available information is not sufficient to predict with confidence the effects of concurrent treatment, especially in patients with compromised left ventricular function or cardiac conduction abnormalities. When introducing such concomitant therapy, care must be taken to monitor blood pressure closely since severe hypotension can occur from the combined effects of the drugs. (See Warnings.)

CONTRAINDICATIONS: Known hypersensitivity reaction to PROCARDIA.

WARNINGS: **Excessive Hypotension:** Although in most patients the hypotensive effect of PROCARDIA is modest and well tolerated, occasional patients have had excessive and poorly tolerated hypotension. These responses have usually occurred during initial titration or at the time of subsequent upward dosage adjustment, and may be more likely in patients on concomitant beta blockers.

Severe hypotension and/or increased fluid volume requirements have been reported in patients receiving PROCARDIA together with a beta blocking agent who underwent coronary artery bypass surgery using high dose fentanyl anesthesia. The interaction with high dose fentanyl appears to be due to the combination of PROCARDIA and a beta blocker, but the possibility that it may occur with PROCARDIA alone, with low doses of fentanyl, in other surgical procedures, or with other narcotic analgesics cannot be ruled out. In PROCARDIA treated patients where surgery using high dose fentanyl anesthesia is contemplated, the physician should be aware of these potential problems and, if the patient's condition permits, sufficient time (at least 36 hours) should be allowed for PROCARDIA to be washed out of the body prior to surgery.

Increased Angina: Occasional patients have developed well documented increased frequency, duration or severity of angina on starting PROCARDIA or at the time of dosage increases. The mechanism of this response is not established but could result from decreased coronary perfusion associated with decreased diastolic pressure with increased heart rate, or from increased demand resulting from increased heart rate alone.

Beta Blocker Withdrawal: Patients recently withdrawn from beta blockers may develop a withdrawal syndrome with increased angina, probably related to increased sensitivity to catecholamines. Initiation of PROCARDIA treatment will not prevent this occurrence and might be expected to exacerbate it by provoking reflex catecholamine release. There have been occasional reports of increased angina in a setting of beta blocker withdrawal and PROCARDIA initiation. It is important to taper beta blockers, if possible, rather than stopping them abruptly before beginning PROCARDIA.

Congestive Heart Failure: Rarely, patients, usually receiving a beta blocker, have developed heart failure after beginning PROCARDIA. Patients with tight aortic stenosis may be at greater risk for such an event.

PRECAUTIONS: **General:** Hypotension. Because PROCARDIA decreases peripheral vascular resistance, careful monitoring of blood pressure during the initial administration and titration of PROCARDIA is suggested. Close observation is especially recommended for patients already taking medications that are known to lower blood pressure. (See Warnings.)

Peripheral edema: Mild to moderate peripheral edema, typically associated with arterial vasodilation and not due to left ventricular dysfunction, occurs in about one in ten patients treated with PROCARDIA. This edema occurs primarily in the lower extremities and usually responds to diuretic therapy. With patients whose angina is complicated by congestive heart failure, care should be taken to differentiate this peripheral edema from the effects of increasing left ventricular dysfunction.

Drug Interactions: Beta-adrenergic blocking agents. (See Indications and Warnings.) Experience in over 1400 patients in a non-comparative clinical trial has shown that concomitant administration of PROCARDIA and beta-blocking agents is usually well tolerated, but there have been occasional literature reports suggesting that the combination may increase the likelihood of congestive heart failure, severe hypotension or exacerbation of angina.

Long acting nitrates. PROCARDIA may be safely co-administered with nitrates, but there have been no controlled studies to evaluate the antianginal effectiveness of this combination.

Digitalis: Administration of PROCARDIA with digoxin increased digoxin levels in nine of twelve normal volunteers. The average increase was 45%. Another investigator found no increase in digoxin levels in thirteen patients with coronary artery disease. In an uncontrolled study of over two hundred patients with congestive heart failure during which digoxin blood levels were not measured, digitalis toxicity was not observed. Since there have been isolated reports of patients with elevated digoxin levels, it is recommended that digoxin levels be monitored when initiating, adjusting, and discontinuing PROCARDIA to avoid possible over- or under-digitalization.

Carcinogenesis, mutagenesis, impairment of fertility: When given to rats prior to mating, nifedipine caused reduced fertility at a dose approximately 30 times the maximum recommended human dose.

Pregnancy, Category C: Please see full prescribing information with reference to teratogenicity in rats, embryotoxicity in rats, mice and rabbits, and abnormalities in monkeys.

ADVERSE REACTIONS: The most common adverse events include dizziness or light-headedness, peripheral edema, nausea, weakness, headache and flushing each occurring in about 10% of patients; transient hypotension in about 5%; palpitation in about 2%; and syncope in about 0.5%. Syncopal episodes did not recur with reduction in the dose of PROCARDIA or concomitant antianginal medication. Additionally, the following have been reported: muscle cramps, nervousness, dyspnea, nasal and chest congestion, diarrhea, constipation, inflammation, joint stiffness, shakiness, sleep disturbances, blurred vision, difficulties in balance, dermatitis, pruritus, urticaria, fever, sweating, chills, and sexual difficulties. Very rarely, introduction of PROCARDIA therapy was associated with an increase in anginal pain, possibly due to associated hypotension.

In addition, more serious adverse events were observed, not readily distinguishable from the natural history of the disease in these patients. It remains possible, however, that some or many of these events were drug related. Myocardial infarction occurred in about 4% of patients and congestive heart failure or pulmonary edema in about 2%. Ventricular arrhythmias or conduction disturbances each occurred in fewer than 0.5% of patients.

Laboratory Tests: Rare, mild to moderate, transient elevations of enzymes such as alkaline phosphatase, CPK, LDH, SGOT and SGPT have been noted, and a single incident of significantly elevated transaminases and alkaline phosphatase was seen in a patient with a history of gall bladder disease after about eleven months of nifedipine therapy. The relationship to PROCARDIA therapy is uncertain. These laboratory abnormalities have rarely been associated with clinical symptoms. Cholestasis, possibly due to PROCARDIA therapy, has been reported twice in the extensive world literature.

HOW SUPPLIED: Each orange, soft gelatin PROCARDIA CAPSULE contains 10 mg of nifedipine. PROCARDIA CAPSULES are supplied in bottles of 100 (NDC 0069 2600 66), 300 (NDC 0069 2600 72), and unit dose (10x10) (NDC 0069 2600 41). The capsules should be protected from light and moisture and stored at controlled room temperature 59 to 77 F (15 to 25 C) in the manufacturer's original container.

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"I have been able to do volunteer work...and feel needed and useful once again."

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Side effects are usually mild (most frequently reported are dizziness or lightheadedness, peripheral edema, nausea, weakness, headache and flushing, each occurring in about 10% of patients, transient hypotension in about 5%, palpitation in about 2% and syncope in about 0.5%).



for the varied faces of angina

* Procardia is indicated for the management of:

- 1) Confirmed vasospastic angina.
- 2) Angina where the clinical presentation suggests a possible vasospastic component.
- 3) Chronic stable angina without evidence of vasospasm in patients who remain symptomatic despite adequate doses of beta blockers and/or nitrates or who cannot tolerate these agents. In chronic stable angina (effort-associated angina) PROCARDIA has been effective in controlled trials of up to eight weeks' duration in reducing angina frequency and increasing exercise tolerance, but confirmation of sustained effectiveness and evaluation of long-term safety in these patients are incomplete.

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Resolved, That local medical societies form a liaison with their county commissions to assist in the development, direction, and evaluation of the local health plans; and be it further

Resolved, That the Kansas Department of Health and Environment and the local health boards be encouraged to inform and involve the health officers in all phases of the health department activities, from planning to the execution of programs.

RESOLUTION NO. 84-26

Long Term Care

WHEREAS, The phrase "long term care" describes a range of medical and supportive services for individuals who have lost some capacity for self-care due to a chronic illness or condition, and who are expected to need care for an extended period; and

WHEREAS, Long term care services are often concerned with minimizing the effects of one or more diseases and delaying deterioration of the person's functional capabilities rather than with curative effects; and

WHEREAS, The projected increases in the numbers and proportion of the elderly requiring long term care will be one of the most critical factors affecting the health care system during the next several decades; and

WHEREAS, An understanding of the key issues and developments in the delivery and financing of care for the aged will help all concerned respond to the changing environment; therefore be it

Resolved, That this 1984 House of Delegates recommend for distribution the statement on long term care which was jointly developed by Kansas Department of Health and Environment; the Kansas Medical Society; Kansas Department on Aging; and Kansas Department of Social and Rehabilitation Services, in addition to other concerned groups, to the following audiences:

- The members of the Kansas Medical Society
- The Governor of Kansas
- Kansas Legislature
- Kansas Statewide Health Coordinating Council
- Kansas Media
- American Medical Association

RESOLUTION NO. 84-27

AMA Public Information Program

WHEREAS, The American Medical Association

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has skillfully and with success represented the best interests of physicians and patients in the U. S.; and

WHEREAS, The forum in which representations are made to Congress in hearings and through lobbying by the AMA is handled with tact and skill such that significant improvement is scarcely to be hoped for; and

WHEREAS, Despite these laudable efforts, contrary testimony in the court of public opinion has steadily eroded many AMA positions and it is manifestly obvious that a public information effort by the AMA — even by paid advertising — is preferable to acceptance of compromise inimical to the best interests of physicians and patients alike; therefore be it

Resolved, That the AMA embark on a campaign calculated to provide the public with information about concerns of doctors regarding, for example, mandatory assignment, the merits and disadvantages of prospective payment, PPOs, "Should I trust my doctor whom I pay and who has everything to gain if I live or the government which can't quite find a politically acceptable way to say it would be cheaper if I died?"; and be it further

Resolved, That these apologetics be presented in the form perhaps of paid essays or ads, if necessary, as in the Mobil Corporation series in *Time Magazine*, but that they achieve maximum feasible public exposure; and be it further

Resolved, That the topics of these essays not be strictly confined to those mentioned, but should include topics deemed in the wisdom of the AMA Board of Trustees to be worthy of public presentation; and be it further

Resolved, That the KMS delegates to the AMA submit this resolution to the AMA annual meeting in June and support it with full vigor.

RESOLUTION NO. 84-28

Commendation for Alex Mitchell, M.D., M.P.H.

WHEREAS, Alex C. Mitchell, M.D., M.P.H. has faithfully served for four years as Medical Director of the Kansas Foundation for Medical Care and has furthered the development of the organization; and

WHEREAS, Alex C. Mitchell, M.D., M.P.H. has been outstanding in the excellent performance of his job; therefore be it

Resolved, That the Kansas Medical Society express its appreciation for Dr. Mitchell's services and dedication; and be it further

Resolved, That Alex C. Mitchell, M.D., M.P.H. be commended for his excellence as Medical Direc-

tor of KFMC, and that this action be communicated to Dr. Mitchell.

RESOLUTION NO. 84-29

The University of Kansas School of Medicine

WHEREAS, A close and fraternal relationship has long existed between the physicians of Kansas and the University of Kansas School of Medicine and its faculty; and

WHEREAS, The community of Edwardsville in Wyandotte County has been declared "medically underserved" in order to justify the establishment of an outpatient clinic by the University; and

WHEREAS, Currently the HMO which was developed by the University is planning a large outpatient facility in the immediate vicinity of a Kansas City, Kansas community hospital; and

WHEREAS, These endeavors are widely believed to be subsidized by the tax monies and facilities of the State of Kansas in direct competition with the private physicians and hospitals of the community; and

WHEREAS, Considerable hostility on the part of the community physicians has been engendered by these actions taken without any consultation or forewarning; therefore be it

Resolved, That the matter of these expanded marketing strategies by the University of Kansas School of Medicine be referred to the KMS Medical School Liaison Committee for open discussion in an attempt to avoid the development of a town-gown schism.

RESOLUTION NO. 84-30

Pre-Admission Hospital Certification

WHEREAS, Wichita physicians have recently been informed by some businesses that their medical care plans now require that all non-emergency hospital admissions of their employees and/or dependents must be authorized in advance by their insurance carrier; and

WHEREAS, This type of utilization review program, when initiated on an isolated basis, has been effective in correcting the practice patterns of specific physicians whose practice patterns are out of line when compared to the practice patterns of other physicians of like training, and

WHEREAS, This type of across the board program creates confusion, disrupts the patient-physician relationship, bypasses local utilization review efforts, and thereby potentially adversely affects patient care; therefore be it

Resolved, That the Kansas Medical Society go on record in opposition to non-emergency pre-admission authorization programs when implemented universally; and be it further

Resolved, That the Kansas Medical Society assist business and industry in establishing workable and effective local utilization and review programs when requested to do so.

RESOLUTION NO. 84-31

Medicare Exit Reviews

WHEREAS, The Kansas Foundation for Medical Care serves as the designated PRO for the State of Kansas and is responsible for carrying out utilization review activities under the Medicare program; and

WHEREAS, The medical profession supports cost containment programs such as utilization review as long as they do not jeopardize or adversely affect patient care; and

WHEREAS, Through the initial exit reviews conducted in two of the Wichita hospitals, 71 cases were decertified and out of this number 52 cases were later recertified at the appeals level; and

WHEREAS, There are no review guidelines for the physician reviewers to follow in performing the exit reviews and no group training nor orientation sessions held for the physician reviewers, leading to inconsistent, subjective and arbitrary decisions, and making the review and appeals process costly, non-productive, and frustrating; therefore be it

Resolved, That the Council of the Kansas Medical Society meet with the representatives of the Kansas Foundation for Medical Care to evaluate the current exit review process; and be it further

Resolved, That the appropriate action be implemented to assure consistency in reviews based on guidelines with adequate physician input to assure that quality patient medical care is not being compromised.

RESOLUTION NO. 84-32

Physician Reimbursement by Third Party Payers

(Not adopted — Referred for study)

WHEREAS, The majority of physician reimbursement is through third party payers; and

WHEREAS, The general public, through its tax dollars, is responsible for financing the majority of all governmental sponsored medical care programs; and

WHEREAS, The established level of payment to physicians under governmental sponsored programs should be considered acceptable to physicians as

being reasonable and payment in full when developed cooperatively by physician fee committees; therefore be it

Resolved, That any alteration of present Blue Shield Common Procedure Coding System (AMA CPT) with the fee schedule, *i.e.*, new coding numbers or numbers to fee change, be accepted by the House of Delegates after review and recommendation by a Fee Committee elected from the House of Delegates.

RESOLUTION NO. 84-33

Freedom of Choice of Primary Care Physician

(Not adopted — Referred for study)

WHEREAS, Third party providers and others in Kansas have traditionally defined pediatricians as well as internists, general surgeons, obstetrician/gynecologists, and family practitioners as primary care physician to certain categories of patients; and

WHEREAS, The recently implemented trial "gatekeeper" concept of delivery of care to Medicaid patients in Sedgwick, Saline, and Ottawa counties excludes some pediatricians, internists, general surgeons, and obstetrician/gynecologists from serving as primary care physicians and will not reimburse them for continuing to provide primary care to their patients; and

WHEREAS, This eliminates freedom of choice of primary care physician for these patients which violates a traditional principle of medical care in Kansas; and

WHEREAS, The elimination of these physicians as primary care physicians would have a significant impact on the availability of primary care to Kansas citizens; therefore be it

Resolved, That the Kansas Medical Society:

- Continue to endorse the freedom of choice by the patient for primary care or other medical service from any physician properly qualified by training and/or experience to deliver such service;
- Endorse the concept that any system of reimbursement of primary care physicians by third parties must include all physicians who are properly qualified to deliver such care even to limited categories of patients; and
- Work with departments of state government and third party carriers to insure that the freedom of choice of qualified primary care physicians will be maintained.

RESOLUTION NO. 84-34

Required Affidavit on Medicare Patients

WHEREAS, Recent regulations require a signed

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affidavit by physicians caring for Medicare recipients that hospital discharge diagnoses not be fraudulent; and

WHEREAS, The required statement threatens the physician with fines and imprisonment; and

WHEREAS, The required affidavit serves no useful purpose in improving quality of care or reducing health care costs; and

WHEREAS, The need for an affidavit implies a lack of integrity on the part of the medical profession; and

WHEREAS, The threat of punishment is demeaning to the physician; therefore be it

Resolved, That the Kansas Medical Society voice a note of strong protest to the appropriate Medicare authority; and be it further

Resolved, That the Kansas Medical Society also notify the Senators and Representatives from Kansas to the United States Congress of its opposition to this affront to professional integrity; and be it further

Resolved, That delegates from the Kansas Medical Society to the American Medical Association initiate or support a similar resolution by that body.

RESOLUTION NO. 84-35

Preferred Insurance Service

Not adopted.

CLASSIFIED ADVERTISEMENTS

Classified advertisements are \$25 per insertion. Copy is limited to six lines. Payment must accompany copy. Deadline is 20th of month preceding month of publication. Box numbers are available at no charge. All advertisements are accepted subject to approval by the Editorial Board.

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RESOLUTION NO. 84-36

Reno County Medical Society and Auxiliary

WHEREAS, The Reno County Medical Society and its Auxiliary have been most generous and gracious hosts for this 125th Annual Meeting of the House of Delegates of the Kansas Medical Society and its Auxiliary; and

WHEREAS, They have provided a most comfortable and congenial setting for the meeting, arranged for stimulating, informative and interesting programs, and for an entertaining and profitable evening for AMAERF; and

WHEREAS, Their efforts have produced an excellent attendance; therefore be it

Resolved, That the Kansas Medical Society here assembled express its thanks and gratitude to the Reno County Medical Society for its efforts in hosting this session of the House of Delegates; and be it further

Resolved, That the KMS express its thanks and gratitude to the Reno County Medical Society Auxiliary for all its work in providing for the comfort, needs, and entertainment of their guests; and be it further

Resolved, That copies of this resolution be sent to the Reno County Medical Society and the Reno County Medical Society Auxiliary.

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References: 1. Kales J et al: *Clin Pharmacol Ther* 12:691-697, Jul-Aug 1971. 2. Kales A et al: *Clin Pharmacol Ther* 18:356-363, Sep 1975. 3. Kales A et al: *Clin Pharmacol Ther* 19:576-583, May 1976. 4. Kales A et al: *Clin Pharmacol Ther* 32:781-788, Dec 1982. 5. Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc* 27:541-546, Dec 1979. 6. Kales A, Kales JD: *J Clin Pharmacol* 3:140-150, Apr 1983. 7. Greenblatt DJ, Allen MD, Shader RI: *Clin Pharmacol Ther* 21:355-361, Mar 1977. 8. Zimmerman AM: *Curr Ther Res* 13:18-22, Jan 1971. 9. Amrein R et al: *Drugs Exp Clin Res* 9(1):85-99, 1983. 10. Monti JM: *Methods Find Exp Clin Pharmacol* 3:303-326, May 1981. 11. Greenblatt DJ et al: *Sleep* 5(Suppl 1):S18-S27, 1982. 12. Kales A et al: *Pharmacology* 26:121-137, 1983.

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Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. An additive effect may occur if alcohol is consumed the day following use for nighttime sedation. This potential may exist for several days following discontinuation. Caution against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Potential impairment of performance of such activities may occur the day following ingestion. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, abrupt discontinuation should be avoided with gradual tapering of dosage for those patients on medication for a prolonged period of time. Use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, light-headedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, e.g., excitement, stimulation and hyperactivity.

Dosage: Individualize for maximum beneficial effect. **Adults:** 30 mg usual dosage; 15 mg may suffice in some patients. **Elderly or debilitated patients:** 15 mg recommended initially until response is determined.

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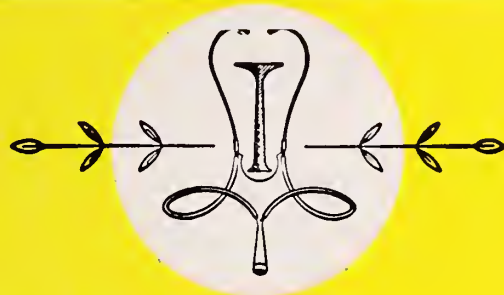
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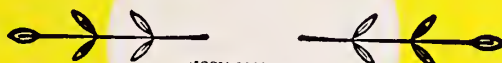
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The JOURNAL of the KANSAS MEDICAL SOCIETY

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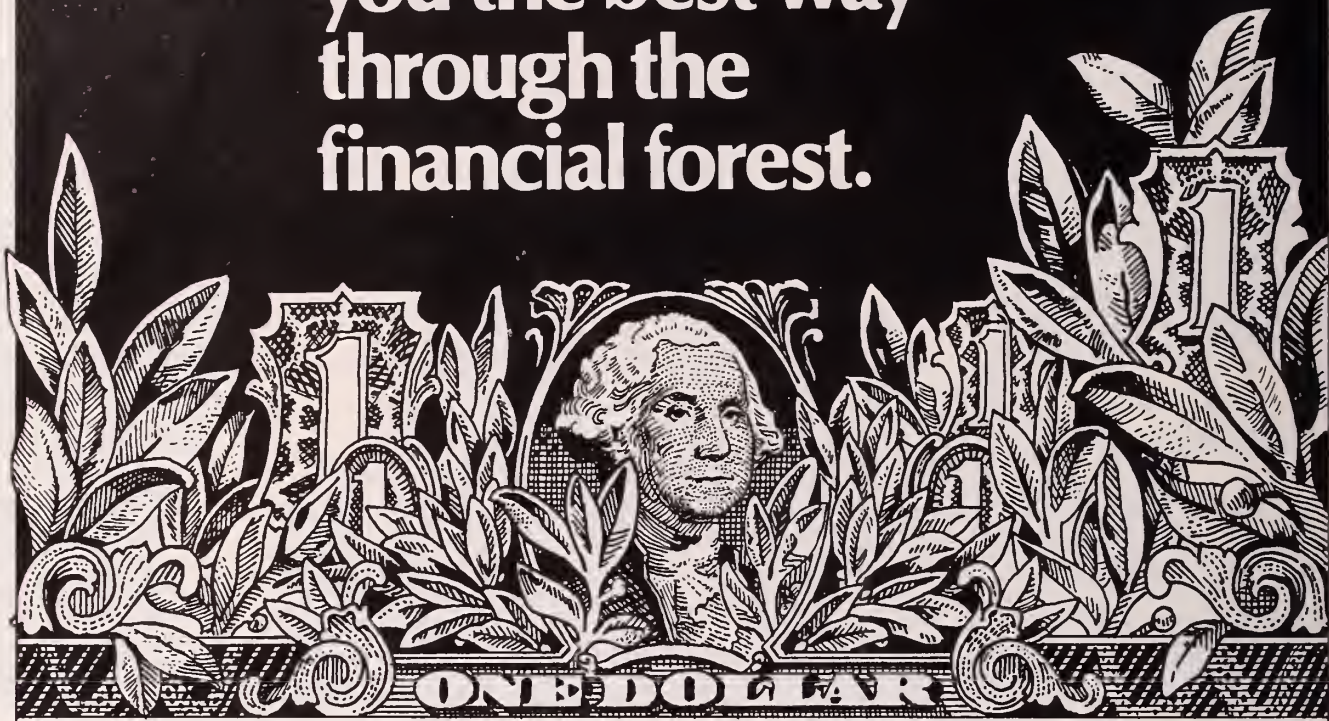
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Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. Associated widened QRS complex or arrhythmia requires prompt additional therapy. Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids, and during concurrent use with amphotericin B or corticosteroids or corticotropin [ACTH]). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Cumulative effects of the drug may develop in patients with impaired renal function. Thiazides should be used with caution in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease. Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic and hemolytic anemia have been reported with thiazides. Thiazides may cause manifestation of latent diabetes mellitus. The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide; dosage adjustments may be necessary. Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported. Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Anti-hypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. Triamterene has been found in renal stones in association with the other usual calculus components. Therefore, 'Dyazide' should be used with caution in patients with histories of stone formation. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide'. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased dietary intake of potassium-rich foods. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function.

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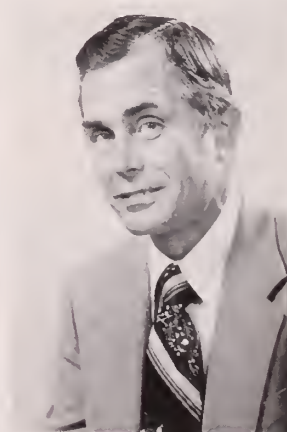
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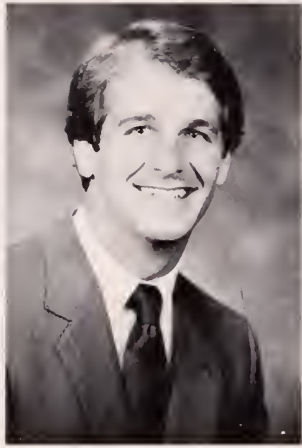


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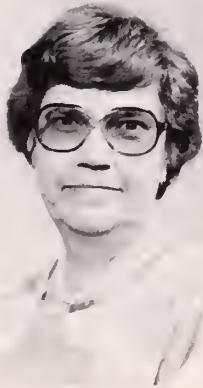
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1. Payroll journal providing monthly totals and division of payroll by type of work performed.
2. Individual earning records indicating the type of work performed. Gross payroll should be totaled by the quarter.
3. Separate record of overtime shown by employee and totaled by class of work for the policy term involved. (Premium for Workers' Compensation is based on straight time pay for all hours worked and does not include $\frac{1}{2}$ extra pay for overtime.) (Not applicable in Delaware, Pennsylvania, and Utah.)
4. Certificates of Workers' Compensation Insurance for all insured sub-contractors.
5. Social Security (Form 941) and State Unemployment Compensation quarterly returns.

Our auditors are instructed to inform you of the date they intend to call on you or to arrange in advance for a convenient time. To assure accurate assignment of your payroll to the proper classes, it is wise for you to arrange to have someone in your organization familiar with employee job assignments available to work with our auditor during the course of the audit.

If your records are kept by an outside accounting firm, make certain the accountants are aware of the impending visit by the auditor so they will have your records available when needed. In the event the accountant is not well informed regarding the duties of various employees, you may wish to brief him in advance of the auditor's visit.

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KANSAS LEGISLATURE

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An added complication... in the treatment of bacterial bronchitis*

Increasing incidence
of ampicillin resistance in
Haemophilus influenzae

Ampicillin Resistant
Haemophilus influenzae

H. influenzae

S. pneumoniae

Brief Summary. Consult the package literature for prescribing information.

Indications and Usage: Ceclor® (cefadroxil, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae* (*Diplococcus pneumoniae*), *Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococcus). Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out.

Precautions: General Precautions—If an allergic reaction to Ceclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of Ceclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Ceclor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with ClinTest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Ceclor. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers—Small amounts of Ceclor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one

Some ampicillin-resistant strains of *Haemophilus influenzae*—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.¹⁻⁶

In clinical trials, patients with bacterial bronchitis due to susceptible strains of *Streptococcus pneumoniae*, *H. influenzae*, *S. pyogenes* (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.⁷

Ceclor®

cefadroxil

Pulvules®, 250 and 500 mg

hour. The effect on nursing infants is not known. Caution should be exercised when Ceclor® (cefadroxil, Lilly) is administered to a nursing woman.

Usage in Children—Safety and effectiveness of this product for use in infants less than one month of age have not been established.

Adverse Reactions: Adverse effects considered related to therapy with Ceclor are uncommon and are listed below.

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100), Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis, arthralgia and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome. Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy. Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations of SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

[061782R]

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.

Note: Ceclor is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

References

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8. Principles and Practice of Infectious Diseases (edited by G. L. Mandell, R. G. Douglas, Jr., and J. E. Bennett), p. 487. New York: John Wiley & Sons, 1979.

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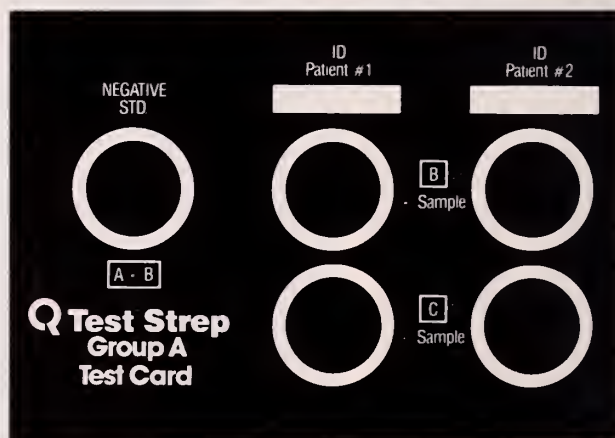
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Parsons — Labette County Medical Center — 316/
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222
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Downs Nursing Center, 1218 Kansas — 913/454-
3329
El Dorado 67042
Bi-County Health Department, Butler County
Courthouse — 316/321-3400
Butler-Greenwood County, 720 W. Central —
316/321-3300

Ellsworth 67439

Ellsworth County, Court House — 913/472-4234

Emporia 66801

Lyon County/Emporia City, 402 Commercial — 316/342-4864

Newman Memorial County Hospital, 12th & Chestnut — 316/343-6800

St. Mary's, 15th & State — 316/342-2450

Fort Scott 66701

Mercy Hospital, 821 Burke — 316/223-2200

Fredonia 66736

Wilson County, 7th & Madison — 316/378-2324

Goodland 67735

Connie's, Route 2, East 8th — 913/899-3147

Sherman County, 1st & Sherman — 913/899-3625

Great Bend 67530

Barton County, 1410 Polk — 316/793-7879

Golden Belt, 3600 Broadway — 316/793-3593

Kansas City

Clinicare Family Health Services, Inc., 510 Southwest Blvd., P.O. Box 3106 66103 — 913/262-6068

Crossland Rehabilitation Agency, 6111 Leavenworth 66104 — 913/334-2005

Catholic Social Services, 229 S. 8th 66101 — 913/621-1504

Visiting Nurse Association, 906 N. 17th 66102 — 913/371-3770

Kingman 67068

Kingman County, Court House — 316/532-2221

Larned 67550

Pawnee County, Court House — 316/285-3866

Lawrence 66044

Douglas County Visiting Nurses Association, 336 Missouri, Suite 201 — 913/843-3738

Leavenworth 66048

Leavenworth City-County Health Department, 422 Walnut — 913/682-0245

Leoti 67861

Wichita County Community, P.O. Box 3 — 316/375-2289

Liberal 67901

Southwest Medical Center, P.O. Box 1340 — 316/624-1651

Lyons 67554

Rice County, Courthouse — 316/257-2359

Manhattan 66502

Manhattan-Riley County, 616 Poyntz — 913/776-4779

Riley County Health Homemaker Services, 219 S. Seth Childs Road — 913/537-0688

Marion 66861

Marion County, 1014 E. Melvin — 316/382-2177

Marysville 66508

Community Memorial Hospital, 708 N. 18th — 913/562-2311

McPherson 67460

McPherson County, 119 N. Maple, P.O. Box 428 — 316/241-1753

Medicine Lodge 67104

Barber County, 710 N. Walnut — 316/886-3294

Minneapolis 67467

Ottawa County, Court House — 913/392-2822

Newton 67114

Harvey County, 8th & Main — 316/283-7232

Norton 67654

P.R.N., East Holme & North Norton — 913/877-2810

Oberlin 67749

Decatur County, 504 N. Penn — 913/475-2222

Oskaloosa 66066

Jefferson County, Court House — 913/863-2447

Oswego 67356

Oswego City Hospital, 900 Barker Drive — 316/795-2921

Ottawa 66067

Franklin County, 13th & S. Main — 316/242-1873

Paola 66071

Miami County, 14 E. Wea — 913/294-2433

Parsons 67357

Labette County, S. 21st, P.O. Box 786 — 316/421-4350

Phillipsburg 67661

Phillips County, Court House — 913/543-2179

Pittsburg 66762

Crawford County, Centennial & Rouse — 316/231-5411

Pratt 67124

Pratt County, 127 S. Howard — 316/672-7436

Sabetha 66534

Nemaha County, 716 S. 11th — 913/284-2288

Salina 67401

Salina-Saline County, 300 W. Ash — 913/827-9376

Shawnee Mission

Always Better Care, Inc., 10111 Santa Fe Drive 66212 — 913/888-4447

Home Health-Home Care, Inc., 8900 State Line, Suite 332 66206 — 913/341-8830

Medical Personnel Pool of Kansas City, 7600 State Line, Suite 200 66208 — 913/341-2181

Stockton 67669

Rooks County, Court House — 913/425-7352

Topeka

Omni-Care Health Systems, Inc., 2930 SW
Wanamaker, Suite 9, Topeka 66610 — 913/
272-0432

Topeka-Shawnee County, 1615 W. 8th 66606 —
913/233-8961

Troy 66087

Doniphan County, Courthouse, P.O. Box 201 —
913/985-3886

Ulysses 67880

Bob Wilson Memorial, 415 N. Main — 316/
356-1266

Washington 66968

Washington County, 115 W. 3rd — 913/325-
2600

Wellington 67152

Sumner County, Court House — 316/326-2774

Westmoreland 66549

Pottawatomie County, 320 Main — 913/457-
3719

Wichita

Agency for Home Health Care of Kansas, 3333 E.
Central, Suite 503 67208 — 316/681-1632

Kansas Masonic Home, 401 S. Seneca 67213 —
316/267-0271

Medical Personnel Pool, 1035 Parklane 67218 —
316/686-3388

Professional Care Associates, 3333 E. Central,
Suite 821 67208 — 316/681-0068

Wesley Care, 550 N. Hillside 67214 — 316/688-
7272

Wichita-Sedgwick County, 1900 E. 9th 67214 —
316/268-8433

Winfield 67156

William Newton Memorial Hospital, 1300 E. 5th
— 316/221-2300

GENETIC COUNSELING CENTERS

Kansas City — Genetic Counseling Center, Division of Medical Genetics, UKSM-KC, 39th & Rainbow Blvd., Kansas City, KS 66103 — 913/588-6043, R. Neil Schimke, M.D., Director; Debra L. Collins, M.S., Genetic Counselor

Topeka — Genetic Counseling Center, 1518 S.W. 8th Street, Topeka, KS 66604 — 913/232-0957, or Kansas City Center

Salina — Asbury Hospital, P.O. Box 1608 — 913/827-9376, or Kansas City Center

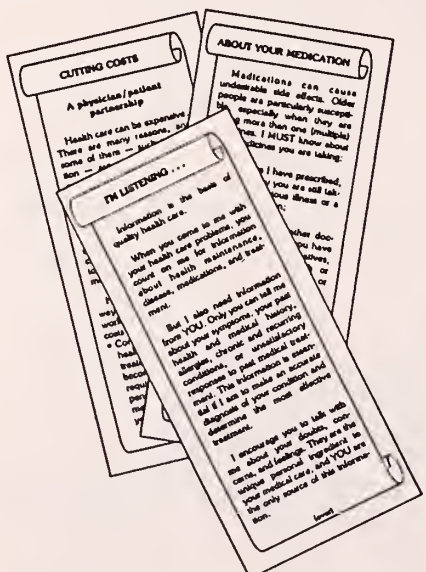
Hays — Post Rock Pediatric Clinic — 913/628-6128, Ext. 29, or Kansas City Center

Parsons — Parsons State Hospital & Training Center — 316/421-6550, Ext. 227, or Kansas City Center

Colby — Citizen's Medical Center — 913/462-7511, Ex. 254, or Kansas City Center

Wichita — Genetic Counseling Unit — Wesley Medical Center, 550 N. Hillside, Wichita, KS 67214 — 316/685-2151, Sechin Cho, M.D., Director

Garden City — Contact Wichita Center



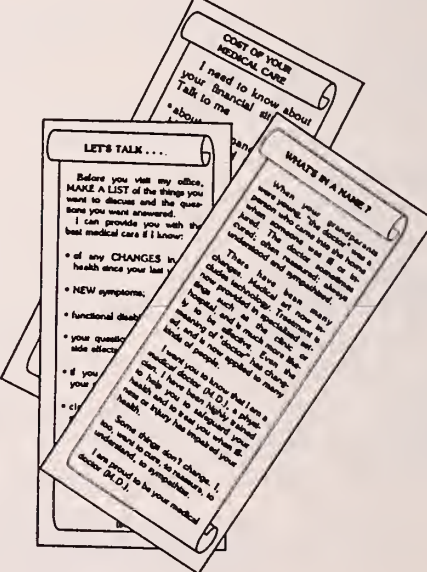
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Principles of MEDICAL ETHICS

Preamble:

The medical profession has long subscribed to a body of ethical statements developed primarily for the benefit of the patient. As a member of this profession, a physician must recognize responsibility not only to patients, but also to society, to other health professionals, and to self. The following Principles adopted by the American Medical Association are not laws, but standards of conduct which define the essentials of honorable behavior for the physician.

- I. A physician shall be dedicated to providing competent medical service with compassion and respect for human dignity.
- II. A physician shall deal honestly with patients and colleagues, and strive to expose those physicians deficient in character or competence, or who engage in fraud or deception.
- III. A physician shall respect the law and also recognize a responsibility to seek changes in those requirements which are contrary to the best interests of the patient.
- IV. A physician shall respect the rights of patients, of colleagues, and of other health professionals, and shall safeguard patient confidences within the constraints of the law.
- V. A physician shall continue to study, apply and advance scientific knowledge, make relevant information available to patients, colleagues, and the public, obtain consultation, and use the talents of other health professionals when indicated.
- VI. A physician shall, in the provision of appropriate patient care, except in emergencies, be free to choose whom to serve, with whom to associate, and the environment in which to provide medical services.
- VII. A physician shall recognize a responsibility to participate in activities contributing to an improved community.

**Adopted by the A.M.A. House of Delegates
July 20-24, 1980**

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0102 University of Alabama School of Medicine, Birmingham
0301 University of Arizona College of Medicine, Tucson
0401 University of Arkansas School of Medicine, Little Rock
0502 University of California School of Medicine, San Francisco
0506 University of Southern California School of Medicine, Los Angeles
0511 Stanford University School of Medicine, Palo Alto
0512 Loma Linda University School of Medicine, Loma Linda—Los Angeles
0515 University of California College of Medicine, Irvine
0702 University of Colorado School of Medicine, Denver
0801 Yale University School of Medicine, New Haven
1001 George Washington University School of Medicine, Washington
1002 Georgetown University School of Medicine, Washington
1003 Howard University College of Medicine, Washington
1102 University of Miami School of Medicine, Miami
1103 University of Florida College of Medicine, Gainesville
1201 Medical College of Georgia, Augusta
1205 Emory University School of Medicine, Atlanta
1601 Rush Medical College, Chicago
1602 University of Chicago Pritzker School of Medicine, Chicago
1604 The Hahnemann Medical College and Hospital, Chicago
1606 Northwestern University Medical School, Chicago
1611 University of Illinois College of Medicine, Chicago
1642 Chicago Medical School University of Health Sciences, Chicago
1643 Loyola University Stritch School of Medicine, Maywood
1676 Chicago College of Osteopathic Medicine, Chicago
1720 Indiana University School of Medicine, Indianapolis
1803 University of Iowa College of Medicine, Iowa City
1875 College of Osteopathic Medicine and Surgery, Des Moines
1902 University of Kansas School of Medicine, Kansas City
2002 University of Louisville School of Medicine, Louisville
2012 University of Kentucky College of Medicine, Lexington
2101 Tulane University School of Medicine, New Orleans
2105 Louisiana State University School of Medicine, New Orleans
2201 Bowdoin Medical School, Brunswick-Portland
2301 University of Maryland School of Medicine, Baltimore
2307 Johns Hopkins University School of Medicine, Baltimore
2401 Harvard Medical School, Boston
2405 Boston University School of Medicine, Boston
2407 Tufts University School of Medicine, Boston
2501 University of Michigan Medical School, Ann Arbor
2507 Wayne State University School of Medicine, Detroit
2512 Michigan State University College of Human Medicine, East Lansing
2604 University of Minnesota Medical School, Minneapolis
2701 University of Mississippi School of Medicine, Jackson
2802 Washington University School of Medicine, St. Louis
2803 University of Missouri School of Medicine, Columbia
2820 University Medical College of Kansas City

- 2822 Ensworth Medical College, St. Joseph
- 2834 St. Louis University School of Medicine, St. Louis
- 2843 Kansas City College of Medicine and Surgery
- 2846 University of Missouri School of Medicine, Kansas City
- 2878 Kansas City College of Osteopathy & Surgery
- 2879 Kirksville College of Osteopathic Medicine, Kirksville
- 3005 University of Nebraska College of Medicine, Omaha
- 3006 Creighton University School of Medicine, Omaha
- 3007 Nebraska College of Medicine, Lincoln
- 3305 College of Medicine & Dentistry of New Jersey — New Jersey Medical School, Newark
- 3401 University of New Mexico School of Medicine, Albuquerque
- 3501 Columbia University College of Physicians and Surgeons, New York
- 3503 Albany Medical College of Union University, Albany
- 3506 State University of New York at Buffalo, School of Medicine, Buffalo
- 3508 State University of New York College of Medicine, Brooklyn
- 3509 New York Medical College, New York
- 3510 Bellevue Hospital Medical College, New York
- 3515 State University of New York College of Medicine, Syracuse
- 3519 New York University School of Medicine, New York
- 3520 Cornell University Medical College, New York
- 3545 University of Rochester School of Medicine and Dentistry, Rochester
- 3546 Albert Einstein College of Medicine of Yeshiva University, New York
- 3601 University of North Carolina School of Medicine, Chapel Hill
- 3605 Bowman Gray School of Medicine, Winston-Salem
- 3607 Duke University School of Medicine, Durham
- 3802 Eclectic Medical College, Cincinnati
- 3806 Case Western Reserve University School of Medicine, Cleveland
- 3840 Ohio State University College of Medicine, Columbus
- 3841 University of Cincinnati College of Medicine, Cincinnati
- 3843 Medical College of Ohio at Toledo, Toledo
- 3901 University of Oklahoma School of Medicine, Oklahoma City
- 3979 Oklahoma College of Osteopathic Medicine and Surgery, Tulsa
- 4002 University of Oregon Medical School, Portland
- 4101 University of Pennsylvania School of Medicine, Philadelphia
- 4102 Jefferson Medical College of Thomas Jefferson University, Philadelphia
- 4107 Medical College of Pennsylvania, Philadelphia
- 4109 Hahnemann Medical College and Hospital, Philadelphia
- 4112 University of Pittsburgh School of Medicine, Pittsburgh
- 4113 Temple University School of Medicine, Philadelphia
- 4114 Pennsylvania State University, Milton S. Hershey Medical Center, Hershey
- 4177 Philadelphia College of Osteopathic Medicine, Philadelphia
- 4201 University of Puerto Rico School of Medicine, San Juan
- 4301 Brown University Division of Biological and Medical Sciences, Providence
- 4501 Medical University of South Carolina College of Medicine, Charleston
- 4705 Vanderbilt University School of Medicine, Nashville
- 4706 University of Tennessee College of Medicine, Memphis
- 4707 Meharry Medical College School of Medicine, Nashville
- 4802 University of Texas Medical Branch, Galveston
- 4804 Baylor College of Medicine, Houston
- 4812 University of Texas Southwestern Medical School, Dallas
- 4813 University of Texas Medical School, San Antonio
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- 4901 University of Utah College of Medicine, Salt Lake City

- 5002 University of Vermont College of Medicine, Burlington
- 5101 University of Virginia School of Medicine, Charlottesville
- 5104 Medical College of Virginia Health Sciences Division of Virginia Commonwealth University, Richmond
- 5107 Eastern Virginia Medical School, Norfolk
- 5404 University of Washington School of Medicine, Seattle
- 5501 West Virginia University School of Medicine, Morgantown
- 5605 University of Wisconsin Medical School, Madison
- 5606 Medical College of Wisconsin, Milwaukee

FOREIGN MEDICAL SCHOOL CODES

CANADA

060 Alberta

- 06001 University of Alberta Faculty of Medicine, Edmonton
- 06002 University of Calgary Faculty of Medicine, Calgary

061 British Columbia

- 06101 University of British Columbia Faculty of Medicine, Vancouver

062 Manitoba

- 06201 University of Manitoba Faculty of Medicine, Winnipeg

065 Ontario

- 06501 University of Toronto Faculty of Medicine, Toronto
- 06505 Queen's University Faculty of Medicine, Kingston

067 Quebec

- 06701 McGill University Faculty of Medicine, Montreal

OTHER FOREIGN

118 Afghanistan

- 11801 Faculty of Medicine, Kabul University, Kabul

132 Argentina

- 13201 Facultad de Ciencias Medicas de la Universidad de Buenos Aires, Buenos Aires
- 13202 Facultad de Ciencias Medicas de la Universidad Nacional de Cordoba, Cordoba
- 13204 Facultad de Ciencias Medicas, Farmacia y Ramos Menores de la Universidad Nacional del Litoral, Rosario, Santa Fe
- 13206 Facultad de Ciencias Medicas de la Universidad Nacional de Cuyo, Mendoza, Mendoza

143 Australia

- 14303 Faculty of Medicine University of Sydney, Sydney, New South Wales

154 Austria

- 15407 Medizinische Fakultat der Universitat Wien, Wien (407-26 from March 13, 1938 to June, 1945)

165 Belgium

- 16501 Faculte de Medecine et de Pharmacie Universite libre de Bruxelles, Bruxelles

176 Bolivia

- 17602 Facultad de Ciencias Medicas de la Universidad Mayor Real y Pontificia de San Francisco Xavier de Chuquisaca, Sucre
- 17603 Facultad de Medicina de la Universidad Mayor de San Simon, Cochabamba

187 Brazil

- 18708 Universidade Federal de Parana, Faculdade de Medicina, Curitiba, Parana

215 Cambodia

- 21501 Ecole Royal de Medicine Du Cambode, Phnompenh

231 Chile

- 23101 Facultad de Medicina de la Universidad de Chile, Santiago

242 China

- 242 China (also see 243 Effective January 1, 1977)
- 24209 St. John's University (Pennsylvania Medical School, Shanghai, Kiangsu (Extinct))
- 24216 National Shanghai Medical College, Shanghai, Kiangsu
- 24217 West China Union University College of Medicine and Dentistry, Chengtu, Szechuan
- 24222 Aurora University Faculty of Medicine, Shanghai, Kiangsu (Extinct)
- 24239 Shansi University Medical College, Taiyuan, Shansi

243 China

- 24338 National Honan University Medical College, Kaifeng, Honan (24238 Prior to 1-17-1)
- 24351 National Defense Medical Center, School of Medicine, Shanghai, Kiangsu (24251 Prior to 1-17-1)

244 Taiwan

- 244 Taiwan (Formosa) effective 1-17-1
- 24402 College of Medicine National Taiwan University, Taipei (38502 Prior to 1-17-1)
- 24404 Taipei Medical College, Taipei (38504 Prior to 11-71)

264 Colombia

- 26401 Facultad de Medicina de la Universidad Nacional de Colombia Ciudad Universitaria, Bogota, Cundinamarca
- 26402 Facultad de Medicina de la Universidad de Cartagena, Cartagena, Bolivar
- 26404 Facultad de Medicina de la Pontificia Universidad Javeriana, Bogota, Cundinamarca
- 26406 Facultad de Medicina de la Universidad de Caldas, Manizales, Caldas
- 26407 Facultad de Medicina de la Universidad del Cauca, Popayan, Cauca

275 Cuba

- 27501 Facultad de Medicina de la Universidad de la Habana, La Habana

286 Czechoslovakia

- 28602 Charles Univerzita Fakulta of PedGen Medicine, Praha

308 Dominican Republic

- 30801 Facultad de Medicina de la Universidad de Santo Domingo, Ciudad, Trujillo

319 Ecuador

- 31901 Facultad de Ciencias Medicas de la Universidad Central, Quito

330 Egypt (United Arab Republic)

- 33002 Kasr-el-Aini Faculty of Medicine Cairo University, Cairo (Formerly Fouad First University Faculty of Medicine)
- 33003 Faculty of Medicine Alexandria University, Alexandria
- 33004 Abbasis Faculty of Medicine, University of Ein Shams, Cairo

341 El Salvador

- 34104 Facultad de Medicina Universidad Nacional del Salvador, San Salvador

352 England

- 35205 School of Medicine University of Leeds, Leeds
- 35207 University of London Faculty of Medicine, London
- 35211 Registrable Qualifications granted by English Conjoint Board (Royal College of Surgeons of England/Royal College of Physicians of London)

385 Formosa (Taiwan)

- 385 (Also see 244 Taiwan [Effective 1-17-1])
- 38501 Kaohsiung (takau) Medical College, Kaohsiung
- 38502 College of Medicine National Taiwan University, Taipei
- 38503 National Defense Medical Center, Taipei
- 38505 China Medical College, Taichung

396 France

- 39606 Faculte de Medecine de l'Universite de Paris, Paris, Seine
- 39607 Faculte mixte de Medecine et de Pharmacie de l'Universite de Toulouse, Toulouse, Haute-Garonne

407 Germany

- 407 Also see 408409—East and West Germany (Effective 1-1-71)
- 40707 Medizinische Fakultät der Georg-August-Universität, Göttingen, Niedersachsen
- 40710 Medizinische Fakultät der Universität Heidelberg, Heidelberg, Baden-Württemberg
- 40715 Medizinische Fakultät der Philipps-Universität, Marburg/Lahn, Hessen
- 40716 Medizinische Fakultät der Ludwig Maximilians-Universität, München, Bayern
- 40721 Medizinische Fakultät der Universität Hamburg, Hamburg
- 40723 Medizinische Fakultät der Johann-Wolfgang-Goethe-Universität, Frankfurt-Am-Main, Hessen
- 40733 Medizinische Fakultät der Freien Universität Berlin, Berlin

409 Germany West

- 40905 Medizinische Fakultät Albert-Ludwigs-Universität Freiburg IM Breisgau
- 40933 Medizinische Fakultät Freien Universität, Berlin, Berlin (40733 Prior to 1-1-71)

418 Greece

- 41801 Faculty of Medicine National University of Athens, Athens
- 41802 Faculty of Medicine University of Thessaloniki, Thessaloniki

429 Guatemala

- 42901 Facultad de Ciencias Medicas, Universidad de San Carlos, Guatemala

451 Honduras

- 45101 Facultad de Medicina y Cirugía de la Universidad Nacional Autónoma de Honduras, Tegucigalpa

473 Hungary

- 47301 Orvosi Fakultás Tudományegyetem, Budapest

495 India (Goa)

- 49501 University of Bombay, Affiliated Medical Colleges are:
 - a. Grant Medical College Bombay University, Bombay, Maharashtra
 - b. Seth Gorhandas Sunderdas Medical College Bombay University, Bombay, Maharashtra
- 49504 Madras Medical College Madras University, Madras, Madras
- 49508 Christian Medical College Punjab University, Ludhiana, Punjab
- 49509 St. John's Medical College, Bangalore, Mysore (before June 1966: Government Medical College, Mysore University, Mysore)
- 49511 Andhra Medical College Andhra University, Visakhapatnam, Andhra
- 49516 Stanley Medical College Madras University, Madras, Madras
- 49518 Assam Medical College Gauhati University, Dibrugarh, Assam
- 49521 Osmania Medical College Osmania University, Hyderabad, Andhra
- 49523 Medical College Baroda University, Baroda, Gujarat
- 49527 Christian Medical College, Vellore, Madras
- 49528 Byramjee Jeejeebhoy Medical College, Poona, Maharashtra
- 49529 Government Medical College Punjab University, Patiala, Punjab
- 49530 Sawai Man Singh Medical College Rajasthan University, Jaipur, Rajasthan
- 49531 Medical College Kerala University, Trivandrum, Kerala
- 49534 Gajra Rao Medical College Vikram University, Gwalior, Madhya Pradesh
- 49535 Karnatak Medical College Karnatak University, Hubli, Mysore
- 49536 All-India Institute of Medical Sciences, New Delhi, Delhi
- 49537 Kasturba Medical College Karnatak University, Manipal, Mysore
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- 49547 Medical College Jabalpur University, Jabalpur, Madhya Pradesh
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- 49554 Rajendra Medical College, Ranchi, Bihar
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- 49568 College Medicine Sciences Banaras Hindu University, Varanasi, Uttar Pradesh
- 49576 Municipal Medical College, Gujarat University, Ahmedabad, Gujarat
- 49596 Lokmanya Tilak Mun Medical College, Bombay University, Bombay, Maharashtra

- 49597 Dr. Vaishampayan Memorial Medical College, Shivaji University, Shalapur, Maharashtra
- 496 India**
49611 Sri Krishna Medical College, Muzaffarpur, Bihar
- 506 Indonesia**
50602 Faculty of Medicine Airlangga Airlangga University, Surabaya
- 517 Iran**
51701 Faculty of Medicine University of Teheran, Teheran
- 528 Iraq**
52801 Faculty of Medicine Baghdad University, Baghdad
- 539 Ireland**
53901 Faculty of Medicine Queen's University of Belfast, Belfast
53902 National University of Ireland, Constituent Colleges are:
a. Faculty of Medicine University College, Dublin
b. Faculty of Medicine University College, Cork
c. Faculty of Medicine, Galway
- 550 Israel**
55001 The Hebrew University-Hadassah Medical School, Jerusalem
- 561 Italy**
56115 Facolta di Medicina e Chirurgia dell'Universita di Perugia, Perugia
56119 Facolta di Medicina e Chirurgia dell'Universita di Siena, Siena
- 572 Japan**
57211 Tokyo Medical College (Nippon Ikadaigaku) Hongo, Tokyo (Extinct)
57241 Faculty of Medicine Shinshu University, Matsumoto, Nagano
57249 Tokyo Medical College, Tokyo
- 583 Korea (South)**
58301 Severence Medical College Yonsei University, Seoul
58302 College of Medicine Seoul National University, Seoul
58303 Korea University Medical College, Seoul
58304 College of Medicine Kyong-Puk National University, Taegu
58306 College of Medicine Chun Nam National University, Kwangju
58309 College of Medicine Pusan National University, Pusan
58310 College of Medicine Catholic University, Seoul
- 605 Lebanon**
60501 Medical School American University of Beirut, Beirut
- 627 Malta**
62701 Faculty of Medicine and Surgery Royal University of Malta, Valetta
- 649 Mexico**
64901 Facultad de Medicina de la Universidad Nacional Autonoma de Mexico, Mexico
64902 Facultad de Medicina de la Universidad de Nuevo Leon, Monterrey, Nuevo Leon
64906 Facultad de Medicina de la Universidad Nacional del Sureste, Merida, Yucatan
64914 Facultad de Medicina de la Universidad Autonoma de Guadalajara, Guadalajara, Jalisco
64936 Centro de Estudios Universidad Xochicalo A.C., Cuernavaca, Morelos
- 660 Netherlands**
66061 Faculteit der Geneeskunde Universiteit Van Amsterdam, Amsterdam
- 671 New Zealand**
67101 Medical School University of Otago, Dunedin
- 704 Pakistan**
70401 King Edward Medical College, Lahore, West Pakistan
70402 Dow Medical College, Karachi, Federal Capital Area
70403 Dacca Medical College, Dacca, East Pakistan
70404 Nishtar Medical College, Multan, West Pakistan
70409 Khyber Medical College, Peshawar, North-West Frontier Province
70410 Chittagong Medical College, Chittagong, East Pakistan (16001 after 7-1-72)

726 Paraguay

72601 Facultad de Medicina de la Universidad Nacional de Asuncion, Asuncion

737 Peru

73701 Facultad de Medicina de San Fernando de la Universidad Nacional Mayor de San Marcos, Lima

73705 Facultad de Medicina de la Universidad Nacional de San Agustin, Arequipa

73706 Facultad de Medicina "Cayetano Heredia" de la Universidad Peruana de Ciencias Medicas y Biologicas, Lima

748 Phillipines

74801 Faculty of Medicine and Surgery University of Santo Tomas, Manila

74802 College of Medicine University of the Phillipines, Manila

74807 College of Medicine Manila Central University, Manila

74808 Institute of Medicine Far Eastern University, Manila

74809 College of Medicine Southwestern University, Cebu City

74810 College of Medicine University of the East, Quezon City

74811 College of Medicine Cebu Institute of Technology, Cebu City

803 Scotland

80301 Faculty of Medicine University of Aberdeen, Aberdeen

80302 University of St. Andrews School of Medicine, Dundee

80303 Faculty of Medicine University of Edinburgh, Edinburgh

836 South Africa

83601 Medical School University of the Witwatersrand, Johannesburg

847 Spain

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84703 Facultad de Medicina de la Universidad de Grenada, Grenada

84704 Facultad de Medicina de la Universidad de Madrid, Madrid

84706 Facultad de Medicina de la Universidad de Zaragoza, Zaragoza

84708 Facultad de Medicina de la Universidad de Valencia, Valencia

84710 Facultad de Medicina de la Universidad de Salamanca, Salamanca

84711 Facultad de Medicine de la Universidad Catolica Navarra, Pamplona

869 Switzerland

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86902 Medizinische Fakultat der Universitat Bern, Bern

86905 Faculte de Medecine de l'Universite de Lausanne, Lausanne

875 Syria

87501 Faculty of Medicine Damascus University, Damascus

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89102 Faculty of Medicine at Sariraj Hospital University of Medical Sciences, Thonburi

89104 Faculty of Medicine at Ramathibodi Hospital, Mahidol University, Bangkok

902 Turkey

90201 Tip Fakultesi Istanbul Universitesi, Istanbul

90205 Hacettepe University Faculty of Medicine, Ankara

913 Union of Soviet Socialist Republics

91302 Voronez Medical Institute, Voronez

941 Viet-Nam South

94101 Faculte mixte de Medicine et de Pharmacie Universite de Saigon, Saigon

957 Yugoslavia

95702 Medicinski Fakultet Univerziteta u Beogradu, Beograd

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 CHEN, TAI-MING, TOPEKA
 CHENG, WEI Y. KANSAS CITY
 CHENOWETH, JOHN M. O.D., OLATHE
 CHERRY JR., ARTHUR C. TOPEKA
 CHERVEN, PHILIP L. HUTCHINSON
 CHI, IL-SUNG, WICHITA
 CHIN, CRAIGHTON, KANSAS CITY
 CHIN, TOM D. KANSAS CITY
 CHO, CHENG T. KANSAS CITY
 CHO, SECHIN, WICHITA
 CHONKO, ARNOLD M. KANSAS CITY
 CHOPRA, RAMAN, WICHITA
 CHOTIMONGKOL, ANUPONG, DOOGUE CITY
 CHOW, STANLEY Y. FORT SCOTT
 CHOY, JAMES K. L. SUN CITY, AZ
 CHRISTENSEN, MARION D. KIOWA
 CHRISTENSEN, SHANE R. KANSAS CITY, MO
 CHRISTIAN, STANLEY J. SHAWNEE MISSION
 CHRISTMAN JR., CARL, WICHITA
 CHRONISTER, BERT, NEODESHA
 CHRYSANT, STEVEN G. KANSAS CITY, MO
 CHUBB, RICHARD M. BAKTER SPRINGS
 CHUNG, JOHN J. SHARON SPRINGS
 CISKEY, WILLIAM J. EUREKA
 CLAASSEN, MILTON A. NEWTON
 CLAASSEN, SAMUEL D. MCPHERSON
 CLARK, CHUCK, KANSAS CITY
 CLARK, COURTNEY, WICHITA
 CLARK, CRAIG M. TOPEKA
 CLARK, DAVID H. SALINA
 CLARK, FRANCIE H. KANSAS CITY
 CLARK, LAURENCE A. WAMEGO
 CLARK, ORVILLE R. ST. PETERSBURG, FL

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 CLARK, ROBERT THOMAS, GUATEMALA CEN. A.
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 CLAY, MICHAEL J. KANSAS CITY, MO
 CLENDENIN, ROBERT KEELE, OLATHE
 CLIFTON, H. DAVID, WICHITA
 CLINE, BYRON W. WICHITA
 CLINTON, DALE L. LAWRENCE
 CLOYD, DAVID W. SHAWNEE MISSION
 CLYMER, DAVID J. SHAWNEE MISSION
 COADY, MARY ANN, KANSAS CITY
 COALE, LLOYD H. KANSAS CITY
 COBB, LESLIE H. MULVANE
 COCHRAN, PAUL W. TOPEKA
 COOY, DOROTHY, HAYS
 COOY, JOHN, HAYS
 COE, RICHARD D. SHAWNEE MISSION
 COFFEY, ROY B. SALINA
 COHEN, JUSTIN THOMAS, WICHITA
 COHEN, LOUIS, TOPEKA
 COHEN, MARC D. SHAWNEE MISSION
 COHEN, ROBERT A. SHAWNEE MISSION
 COHLMIA, JERRY B. WICHITA
 COHY, STEVEN G. SHAWNEE MISSION
 COHNBERG, ROSELLEN E. CEDAR VALE
 COKELEY, JOHN M. TOPEKA
 COCKER, W. LAURENCE, TOPEKA
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 COLEMAN, ROBERT L. SHAWNEE MISSION
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 COLLINS, EDWARD JOSEPH, TOPEKA
 COLLINS, ELISABETH B. TOPEKA
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 COLLINS, SHARON A. GARDEN CITY
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 COMBS, PETER S. LEAVENWORTH
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 CONARD, CLAIR C. DOOGUE CITY
 CONCANNON, CRAIG A. WICHITA
 CONCEPCION JR., EUGENIO S. WICHITA
 CONVELLY, MAURICE R. SALINA
 CONNER, BRIAN, SALINA
 CONRADOY, PETER A. WICHITA
 CONROY, ROBERT W. TOPEKA
 COOK, DONALD RAY, WICHITA
 COOK, G. EDWARD, WICHITA
 COOK, JAMES O. KANSAS CITY
 COOK, THEODORE R. KANSAS CITY
 COOK, ALLAN R. KANSAS CITY
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 COONFIELD, JAMES W. KANSAS CITY
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 CORBIN, MURRAY D. SHAWNEE MISSION
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 CORDRY, VINCEL R. O.D., WICHITA
 CORNELL, EARL G. CONCORDIA
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 COSSMAN, F. PRICE, WICHITA
 COTTON, ROBERT T. TOPEKA
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 COULTER, THOMAS B. SHAWNEE MISSION
 COULTER, THAYNE A. O.D., CLOYE
 COVERT, THOMAS J. SALINA
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 CRAMM, RUSSELL E. HAYS
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 CRANE, DAVID O. WICHITA
 CRARY, JOHN E. TOPEKA
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CZAPANSKY, DESIREE K. KANSAS CITY

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DANIEL, ROBERT M. VALLEY CENTER
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DIAZ, SALVADOR F. WICHITA
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DICK, WILLIS G. IDLA
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DICKINSON, CHARLES R. COFFEYVILLE
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DIXON, RAYMOND W. COFFEYVILLE
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DONATELLE, EDWARD P. WICHITA
DONLEY, JAMES L. WICHITA
DONNELL, JAMES F. WICHITA
DONNELL, JAMES M. WICHITA
DONNELLY, WILLIAM P. GARDEN CITY
DOORNBOS, J. FRED, WICHITA
DOORSCH, JOHN N. HAYS
DOUBEK, DEBBIE L. KANSAS CITY
DOUBEK, HERBERT D. BELLEVILLE
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DOUGHERTY JR., THOMAS M. P. WICHITA
DOUGHERTY, DOUGLAS DAVID, WICHITA
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DDZIER, FRED S. HERINGTON
DRAEMEL, H. RICHARD, SALINA
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DRAZEK, GEORGE, WICHITA
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DUNLAP, MICHAEL D. ATCHISON
DUNLAP, RICHARD L. LAWRENCE
DUNN, DANIEL R. SCOTT CITY
DUNN, MARVIN I. KANSAS CITY
DUNSHEE, CARLYLE M. FORT SCOTT
DUNSHEE, CHERYL A. WICHITA
DURAND, ANTONIO C. WICHITA
DURKEE, WILLIAM R. MANHATTAN
DURST JR., ROBERT D. TOPEKA
DUYSAK, SAMI. LEAVENWORTH
DYCK, ARTHUR H. MCPHERSON
DYCK, ERIC LEE, HAYS
DYCK, GEORGE, NEWTON
DYER, VERNON E. WICHITA
DYSART, JACK C. STERLING

E

EASTES, GARY DEAN, HALSTEAD
EATON, EDWARD L. TOPEKA
EATON, GLEN E. SALINA
EATON, LESLIE F. SALINA
ECK, MARCI J. WICHITA
ECKART, DE MEZLE E. HUTCHINSON
ECKERT, WILLIAM G. WICHITA
EDDY, VICTOR M. HAYS
EDRZDZ, M. LUZ LUNA, COFFEYVILLE
EDWARDS, DAVID J. EMPORIA
EDWARDS, MANIS C. WICHITA
EDWARDS, SHELLEY J. KANSAS CITY
EGBERT, ANNE MARSH, WICHITA
EGEA, FERNANDO M. KANSAS CITY
EGELHOF, RICHARD H. WICHITA
EICHORN, FRANK D. GARDEN CITY
EIDT, DAVID W. OLATHE
EIKERMAN, WILLIAM C. SHAWNEE MISSION
EISEMANN, ALLAN D. KANSAS CITY
ELANGOVAN, SUDHA, WICHITA
ELDER, DOUGLAS M. TOPEKA
ELLIS, BOBBY J. EMPORIA
ELLIS, HARVEY D. WICHITA
ELLIS, JOSEPH G. WICHITA
ELLIS, LAVELLE A. KANSAS CITY
ELLISON, PAUL D. SALINA
ELNEN, WALTER T. WICHITA
EMPSON, CHARLES L. INDEPENDENCE
ENDERS, WRAY, SHAWNEE MISSION
ENNS, EUGENE K. NEWTON
ENNS, JAMES H. LAKE HAVASU CITY, AZ
ENDICH, RONALD, WICHITA
ENS, GERHARD GEORGE, HILLSBORO
ENS, PETER, HILLSBORO
EPLEE, JOHN R. ATCHISON
ERBACHER, GEORGE E. D.D., ATWOOD
ERICKSON, CLARENCE W. PITTSBURG
ERKEN, RONALD V. WICHITA
ERNST, R. L. WICHITA
ERNST, TARI VAE, WICHITA
ESCH, JOHN G. PITTSBURG
ESRIG, HAROLD L. D.D., SHAWNEE MISSION
ESTEP, THOMAS H. WICHITA
ESTRADA, EDMUND C. LIBERAL
ESTRADA, LINA, LIBERAL
ETZENHUSER III, RUSSELL D. SHAWNEE MISSION
EVANS, CAROL ANN, SHAWNEE MISSION
EVANS, FARRIS D. WICHITA
EVANS, GRANT E. WICHITA
EVANS, JOHN F. WICHITA
EVANS, RICHARD W. WICHITA
EVANS, ROGER WILLIAMS, WICHITA
EVANS, WILLIAM R. GREAT BEND
EVANS JR., WILLIAM E. SHAWNEE MISSION
EWING, THOMAS D. LARNED
EYSTER, ROBERT L. WICHITA

F

FABIAN, CARL J. KANSAS CITY
FAIDLEY, CHERYL K. KANSAS CITY
FAILING, TRENT L. OLATHE
FAIRCHILD, JOHN A. MANHATTAN
FAIRCHILD, RICHARD S. TOPEKA
FAIRMAN, DAN S. KANSAS CITY, MO
FALLON, JOHN H. KANSAS CITY, MO
FALTER, RICHARD T. HUTCHINSON
FARHA, GEORGE J. WICHITA
FARHA, S. JIM, WICHITA
FARLEY, JAMES A. WICHITA
FARRIS, RONNIE S. EMPORIA
FAST, ROBERT E. ATCHISON
FAST, W. SPENCER, ATCHISON
FEAGAN, JERRY, TOPEKA
FEIGHNY, ROBERT E. SALINA
FELDMAYER, SEELEY T. MEADE
FENDER JR., THOMAS H. WICHITA

FENT, LEE S. NEWTON
FENTON, ROBERT M. GARDEN CITY
FERGUSON, ROBERT LEON, SHAWNEE MISSION
FERNANDEZ, HECTOR D. HOISINGTON
FERNANDEZ, LUIS A. TOPEKA
FERNIE, ROBERT W. BOULDER, CO
FERREE, RICHARD ALLAN, MCPHERSON
FERRELL, DONALD P. WICHITA
FERRERI, ROGER N. WICHITA
FERRIS, BRUCE G. WICHITA
FESTOFF, BARRY W. KANSAS CITY, MO
FEUILLE JR., EDMOND G. WICHITA
FEVURLY, CHRIS D. LEAVENWORTH
FIELD, RICHARD A. TOPEKA
FIELDS, GALEN W. MCPHERSON
FIELDS, STEPHEN, D.D., WICHITA
FIESER, CARL W. GREAT BEND
FILLEY, VERNON W. PRATT
FILLMAN, ELDON W. TOPEKA
FINK, ABRAHAM A. PLANTATION, FL
FINLEY, DENNIS R. WICHITA
FISCHER, REX R. MANHATTAN
FISHER, JAMES B. WICHITA
FISHER, RAY F. WICHITA
FISHER, RONALD M. SCOTLAND
FITZGERALD, EDWARD J. WICHITA
FITZIG, SANFORD, WICHITA
FITZPATRICK, M. ROBERT, KANSAS CITY
FLANDERS, M. ALDEN, EDINBURGH, TX
FLANNER, FRANK R. HOISINGTON
FLECKENSTEIN, CHARLES S. OMAGA
FLEMING, FJAVEY W. WICHITA
FLOERSCH, HUBERT M. KANSAS CITY
FLOERSCH JR., CLELL B. WICHITA
FLUTER, GEORGE G. KANSAS CITY
FORD, CHARLES R. WICHITA
FORD, FRED L. TOPEKA
FORDYCE, NORMAN, SHAWNEE MISSION
FOREY, JOHN D. KANSAS CITY
FORSTER JR., LOUIS G. SALINA
FORTUNE, CEDRIC B. OLATHE
FOSS, DANIEL C. HUTCHINSON
FOSTER, D. BERNARD, TOPEKA
FOULDER, DENNIS L. WINFIELD
FOULDER, ROBERT J. WICHITA
FOULDER, WAYNE L. CONCORDIA
FOX, DEANNA K. KANSAS CITY
FOX, HOWARD A. KANSAS CITY
FOX, REBECCA R. KANSAS CITY
FRANCIS, ANTHONY E. SALINA
FRANCIS, NORTON L. WICHITA
FRANCISCO, CLARENCE L. SHAWNEE MISSION
FRANCISCO, DAN A. WICHITA
FRANCISCO, EDGARDO, HORTON
FRANCISCO, LINDA L. WICHITA
FRANCISCO, W. DAVID, KANSAS CITY
FRANKLIN JR., BENJAMIN A. TOPEKA
FRANSEN, HERBERT, NEWTON
FRANSEN, PAUL H. NEWTON
FRANTZ, SHIRLEY J. WICHITA
FRANZ, ROBERT G. HILLSBORO
FRAZIER, RICHARD L. EMPORIA
FRETCHETTE, ALAN R. KANSAS CITY, MO
FREDERICK, M. F. HUGSTON
FREDRICKSON, DOREN D. KANSAS CITY
FREDRICKSON, DUANE E. LINDSBORG
FREDERSON JR., WARREN S. CLYDE
FREEMAN, F. GILES, PRATT
FREEMAN, FRED A. MANHATTAN
FREEMAN, MALCOLM C. PITTSBURG
FREEMAN, RAYMOND S. SALINA
FRENCH, JAMES E. WICHITA
FRENCH, JEROME E. WICHITA
FRIESEN, DALE, LAWRENCE
FRIESEN, FLORENCE V. HESSTON
FRIESEN, ORLANDO J. BUHLER
FRIESEN, STANLEY R. KANSAS CITY
FRIGGERI, ROBERT W. GIRARD
FRITZ, GEORGE E. KEWADIN, MI
FRITZMEIER, WILLIAM H. WICHITA
FROMER, JOEL, WICHITA
FROMY, ARTHUR H. WICHITA
FRY, LUTHER L. GARDEN CITY
FUGATE, CARL L. WICHITA
FULLER, JERYL G. SHAWNEE MISSION
FULLER, DEYLD D. LINDSBORG
FULTON, JOHN K. WICHITA
FUNK, EDWARD D. BDNNER SPRINGS

G

GABRIELLI JR., WILLIAM F. KANSAS CITY
GALBUT, ALAN S. SHAWNEE MISSION
GALICIA, JOSEPH P. WICHITA
GALLER, GREG WAYNE, KANSAS CITY
GALLIGAN, JANET M. S. PORTLAND, ME
GALVAN, ALONSO, WICHITA
GANDHI, SHANTIKUMAR K. TOPEKA
GANN, E. LAMONTE, EMPORIA
GANS, FREDERICK A. SALINA
GANZARAIN, RAMON C. TOPEKA
GARCIA, FRANCISCO, SHAWNEE MISSION
GARCIA, GOJLD C. EMPORIA
GARCIA, GUILLERMO D. DODGE CITY
GARCIA-FERRER, CIRA M. SHAWNEE MISSION
GARD, RAYMOND F. PRATT
GARD, RICHARD A. HUTCHINSON
GARDINER, ROBERT C. SHAWNEE MISSION

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 GARDNER, JAMES D. MANHATTAN
 GAREY, WILLIAM JOHN. CHANUTE
 GATEND, JOSEPH. GREAT BEND
 GATSCHE, TIMOTHY P. WICHITA
 GAUGHAN, MICHAEL J. SHAWNEE MISSION
 GAY, JOHN D. TOPEKA
 GEHRT, EARL B. CHANUTE
 GEITZ, JAMES M. EMPORIA
 GELVIN, E. RAYMOND, CONCORDIA
 GENCH, RAYMOND L. CARMEL, CA
 GENOEL, JOSEPH E. TOPEKA
 GENILO, ANANCIO C. WICHITA
 GENILO, CELESTE A. WICHITA
 GENTRY, KALE C. SHAWNEE MISSION
 GEORGE, EARL F. WICHITA
 GEORGE, M. DON. WICHITA
 GERBER, ALLEN D. WICHITA
 GERBER, HARRY A. LEAVENWORTH
 GERJARUSAK, JAPAS, KANSAS CITY
 GERROLD, LINDA L. SHAWNEE MISSION
 GESSLER, DONALD J. HOUSTON, TX
 GETTLER, DEAN T. FORT SCOTT
 GIBBS, EUGENE, COFFEYVILLE
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 GIMPLE, KENNETH, TOPEKA
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 GLATTER, THOMAS R. KANSAS CITY, MO
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 GOERING, EMIL L. BURLINGAME
 GOERING, RANDALL V. WICHITA
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 GOLDBERG, JOHN M. KANSAS CITY
 GOLDSTEIN, ALAN D. KANSAS CITY, MO
 GOLDSTEIN, GERALD L. SHAWNEE MISSION
 GOLLERKERI, MOHAN P. SHAWNEE MISSION
 GOLLIER, ROBERT A. OTTAWA
 GOLLIER II, ROBERT A. OTTAWA
 GOMETZ, MODESTO S. PITTSBURG
 GOMEZ, FRANCISCO, SHAWNEE MISSION
 GONTERO, ELIZABETH K. KANSAS CITY
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 GOOD, MICHAEL W. CLAY CENTER
 GOOD, WENDELL LITSL, SHAWNEE MISSION
 GOODPASTURE, HEWITT C. WICHITA
 GOODPASTURE, WILLARD C. HUTCHINSON
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 GOTO, HIROSHI, KANSAS CITY
 GOYLE, KRISHAN K. WICHITA
 GOYLE, VIMAL, WICHITA
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 GRABER, CHARLES, NEWTON
 GRABER, KAROLYN M. KANSAS CITY
 GRADY, KENNETH L. KANSAS CITY
 GRAHAM, KENNETH L. LEAVENWORTH
 GRAHAM, SUSAN B. SHAWNEE MISSION
 GRAHAM, THOMAS W. LEAVENWORTH
 GRAHAM JR, CHARLES P. TOPEKA
 GRANT, MICHAEL D. CONCORDIA
 GRANTMAN, JARED J. KANSAS CITY
 GRASHOFF, JOYCE A. SHAWNEE MISSION
 GRAUEL, CHARLES W. WICHITA
 GRAVES, JACK W. WICHITA
 GRAVES, KATHRYN, HUTCHINSON
 GRAY, C. K. LAWRENCE
 GRAY, C. LUCIEN, WICHITA
 GRAY, DAVID E. TOPEKA
 GRAY, H. TOM, WICHITA
 GRAY, SCOTT E. LAWRENCE
 GRAYB, ANTOINE S. TOPEKA

GREEN, LAWRENCE C. ARKANSAS CITY
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 GREENBERGER, N. J. KANSAS CITY
 GREENE, HORACE T. TOPEKA
 GREENE, LAWRENCE S. SHAWNEE MISSION
 GREENE, RUSSELL E. TOPEKA
 GREENWOOD, EDWARD O. TOPEKA
 GREER, JAMES F. GARDEN CITY
 GREER, JAMES A. WICHITA
 GREER, RICHARD H. TOPEKA
 GREILINGER, BART A. KANSAS CITY
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 GUTHRIE, RICHARD A. WICHITA
 GUTOVITZ, ALLEN LOUIS, TOPEKA
 GUZMAN, MANUEL, SALINA
 *WINN, DOUGLAS R. WICHITA

H

HA, SANG W. COFFEYVILLE
 HABASHY, SHAWKY V. F. WICHITA
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 HACKER, ELAINE MARY, TOPEKA
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 HAEFFNER, WILLIAM N. EL DORADO
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 HSU, CHENG H, TOPEKA
 HSU, SHIN-FU, TOPEKA
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 HUANG, JONSON, TOPEKA
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 HUTCHISON, MARC K, HAYS
 HUTSEY, PAUL J, WICHITA
 HUTTON, FREDERICK A, TOPEKA
 HWA, EUGENE C, NEWTON
 HYLAND, JOSEPH M, TOPEKA
 HYMAN, HENRY T, D.O., KANSAS CITY, MO
 HYNES, HENRY E, WICHITA

I

IBARRA, J LUIS, WICHITA
 IBARRA, RICHARD C, KANSAS CITY
 IOBE, S. BAOR, WICHITA
 IGLINSKY, W L, HALSTEAD
 IHRIQ, ROGER W, HUGOTON
 ILIFF, R DOUGLAS, TOPEKA
 ILORETA, ALFREDO T, TOPEKA
 INGHAM JR, M LAIRD, LAWRENCE
 INGRAM, JOHN E, KANSAS CITY
 INNES, ROBERT C, SHAWNEE MISSION
 IRBY, ADDISON C, FORT SCOTT
 IRBY, PRATT, FORT SCOTT
 IRWIN, RICHARD L, NEWTON
 ISAAC, CHARLES A, NEWTON
 *ISAACS, JUANITA J, WICHITA
 ISAACSON, RICHARD M, TOPEKA
 ITURRALDE, GEORGE, SHAWNEE MISSION
 IWAY, BELINDA, ELKHART
 IWAY, OLIVIA N, ELKHART

J

JABEL, JUVENAL T, SATANTA
 JACKS, J WARREN, HAWAEE, AR
 JACKSON, CHARLES R, WICHITA
 JACKSON, LINDA H, TOPEKA
 JACKSON, MICHAEL D, GARDEN CITY
 JACKSON, ROBERT V, SHAWNEE MISSION
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 JACKSON JR, DELMAS A, SALINA
 JACOB, KANNAMPALLY L, EL DORADO
 JACOBS, DANIEL H, KANSAS CITY
 JACOBS, DAVID S, KANSAS CITY
 JACOBS, RAE R, KANSAS CITY
 JACOBSEN, OWIGHT SKINNER, COLBY
 JACOBY II, ROBERT E, TOPEKA
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JAMES, VERNON L, WICHITA
 JAMES, DONALD R, SHAWNEE MISSION
 JANSSEN, ERWIN T, TOPEKA
 JARROTT, JOHN D, HUTCHINSON
 JAWADI, JAMEELA MUSAIN, WICHITA
 JAYARAM, MARANDAPALLI R, KANSAS CITY
 JAZAYERLI, NABIL, WICHITA
 JEHAN, SAYED S, WICHITA
 JENNEY, CHARLES B, WICHITA
 JENSEN, DARAN L, WICHITA
 JENSEN, THOMAS M, OLATHE
 JESTER, SHELBY L, WICHITA
 JEWELL, ROSS L, ST FRANCIS
 JEWELL, WILLIAM R, KANSAS CITY
 JIRICKO, WILDS, COFFEYVILLE
 JOHLER, TERRY HARTWIG, LEAVENWORTH
 JOHNSON, BRUCE E, KANSAS CITY
 JOHNSON, CAROL ANN, WICHITA
 JOHNSON, CYNDA A, KANSAS CITY
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 JOHNSON, RANDOL C, HUTCHINSON
 JOHNSON, RICHARD L, HUTCHINSON
 JOHNSON, TERESA F, KANSAS CITY, MO
 JOHNSON, THOMAS E, WICHITA
 JOHNSTON, JAN M, KANSAS CITY, MO
 JONES, CHARLES E, SHAWNEE MISSION
 JONES, DAVID B, SHAWNEE MISSION
 JONES, EDWARD L, GREAT BEND
 JONES, FORREST H, COLUMBUS
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 JOSEPH, BRIAN W, TOPEKA
 JOSEPH, HOWARD F, LAWRENCE
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 JOUVENAT, NEIL C, SHAWNEE MISSION
 JOYCE, G BERNARD, TOPEKA
 JJBELT, HILBERT P, MANHATTAN
 JUOILLA JR, FRANCISCO, WICHITA
 JUSTUS, WILLIAM J, PLEASANTON

K

KADIAN, RAJESH S, SHAWNEE MISSION
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 KAROATZKE, E STANLEY, WICHITA
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 KARLIN, CHARLES A, SAN DIEGO, CA
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 KERR, GERALD F, FORT SCOTT
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 KHOURY, GEORGE H, WICHITA
 KHURANA, SATISH K, SHAWNEE MISSION
 KIFER, C JAMES, HAYS
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 KIM, JONG M, KANSAS CITY
 KIM, PAIK N, WICHITA
 KIM, SUCHA, KANSAS CITY
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 KIMBALL, RICHARD R, MANKATO
 KIMBLE, JAMES A, WICHITA
 KIMMEL, KENNETH K, HALSTEAD
 KIMURA, CHARLES C, SHAWNEE MISSION
 KIMURA, STEPHEN H, SHAWNEE MISSION
 KINOLING, PAUL H, TOPEKA
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 KINPORTS SR, EDWARD B, KANSAS CITY, MO
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 KIRCHNER, FERNANDO R, KANSAS CITY
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 KIRK JR, E DAVID, WICHITA
 KIRKEGAARD, RODGER S, TOPEKA
 KISER, JOHN L, WICHITA
 KISER, WILLARD J, WICHITA
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 KISHORE, SHEELA, PARSONS
 KITCHEN, ROBERT R, WICHITA
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 KLENDIA JR, MARTIN B, BELOIT
 KLEWER, VERNON L, NEWTON
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 KLINGMAN, DIANE D, ANDOVER
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 KNIGHT, LAURA C, WICHITA
 KNIGHT, PHILIP J, WICHITA
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 KOKSAL, TOM, GARDEN CITY
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 KROLL, HARRY G, TOPEKA

L

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 LATIMER, KATHERINE, WICHITA
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 LEE, KYD R, KANSAS CITY
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 LEGASPI JR, PEDRO L, SHAWNEE MISSION
 LEGER, LEE H, FT MYERS, FL
 LEGLER, GARY LEE, D.D., TOPEKA
 LEIFER, WILLIAM N, TOPEKA
 LEIKER, JOSEPH, ARKANSAS CITY
 LEISY, JERALD W, WICHITA
 LEITCH, DAVID A, GARNETT
 LEITNER, YORAM B, WICHITA
 LENDINE JR, ALBERT N, KANSAS CITY
 LENEVE, ROBERT T, HUGOTON
 LENSKE JR, FRANCIS K, IOLA
 LENTZ, WILLIAM R, TOPEKA
 LED, WILLIAM A, KANSAS CITY
 LESSENDEN, GLENN A, LAWRENCE
 LESSENDEN JR, C M, TOPEKA
 LESSER, DAN E, HUTCHINSON
 LESSIN, DIANNA L, KANSAS CITY, MO
 LESTER, JOHN BUCKLES, SHAWNEE MISSION
 LETTNER, HANS T, HUTCHINSON
 LEVINE, ERROL, KANSAS CITY
 LEVINE, WILLIAM R, WICHITA
 LEVY, EDWIN Z, TOPEKA
 LEWIN, WALTER, SHAWNEE MISSION
 LEWIS, JAMES E, SHAWNEE MISSION
 LEWIS JR, H DANIEL, KANSAS CITY, MO
 LIBEL, ROY, KANSAS CITY
 LICHTENMAN, JOHN B, KANSAS CITY, MO
 LIEBERMAN, BRUCE IRWIN, KANSAS CITY
 LIES, BARTHEL N, COLWICH
 LIES, RICHARD B, WICHITA
 LIESMANN, GEORGE E, TOPEKA
 LIESMANN, JEAN ELIZABETH, TOPEKA
 LILICH, MAUREEN A, KANSAS CITY, MO
 LIN, JOE J, WICHITA
 LIND II, EDWARD J, GARDEN PLAIN
 LINDHOLM, GERALD R, NEWTON
 LINDSLEY, CAROL B, KANSAS CITY
 LINDSLEY, HERBERT B, KANSAS CITY
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 LINHART, RONALD D, WICHITA
 LINN, CATHERINE P, KANSAS CITY
 LINSLEY, MICHAEL A, KANSAS CITY
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 LIPSEY, JAMES H, SHAWNEE MISSION
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 LITTLE, L GILBERT, WICHITA
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 LOEWY, HENRY H, WICHITA
 LOEWY, PETER S, MILLSBORO
 LOEWY, WILLIAM C, WICHITA
 LOGAN, GEDFREY G, WICHITA
 LOGANBELL, WARREN J, MOUNDRIDGE
 LOHMEYER, KENNETH L, GREEN VALLEY, AZ
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 LOKER, JAMES L, SHAWNEE MISSION
 LONEY, JOHN M, BELDIT
 LONG, EDWARD E, HUMBOLDT
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 LONG, ROBERT C, NORTON
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 LOSEE, JOHN M, WICHITA
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 LOVE, ROBERT H, WICHITA
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 MANAHAN, G EUGENE, LAWRENCE
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 MANGOLD, JOEL VOYCE, KANSAS CITY
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 MARSHALL, GEORGE W, SALINA
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 MARTIN, OLIVER L, SALINA
 MARTIN, RONALD L, KANSAS CITY
 MARTIN, WILLIAM D, TOPEKA
 MARTIN JR, GLEN E, WICHITA
 *MARTINAK, JOSEPH F, TOPEKA
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 MELEAN, JAIME, WICHITA
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 MENDIONES, L MARLENE, WICHITA
 MENDIONES, RUPERTO D, WICHITA
 MENDLICK, R MICHAEL, OLATHE
 MENEHAN, H JAMES, WICHITA
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 MENNINGER, W WALTER, TOPEKA
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 MERRITT, JOE P, WICHITA
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 MERSON, JAMES C, WICHITA
 MESINA, ROLAND R, KANSAS CITY
 MESROPIAN, GEORGE D, ATCHISON
 MESSANDRE, DEBRA L, WICHITA
 MEYER, JACK R, NORTON
 MEYER, MARK E, TOPEKA
 MEYER, D WARREN, TOPEKA
 MEYER, WARREN E, WICHITA
 MEYERS, STEPHEN, GARDEN CITY
 MICHELBACH, ALBERT P, WICHITA
 MIGUELINO, OLIVER M, EMPORIA
 MIM, ALEXANDER, CHANUTE
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 MILLER, ABRAHAM H, MANHATTAN
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 MILLER, DEAN M, PARSONS
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 MILLER, FREEMAN LANCE, SHAWNEE MISSION
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 MILLER, KEVIN E, KANSAS CITY
 MILLER, LAWRENCE H, DERBY
 MILLER, MONTY B, SCOTT AFB, IL
 MILLER, PHILIP A, PHOENIX, AZ
 MILLER, ROBERT E, GARDEN CITY
 *MILLER, ROGER M, DERBY
 MILLER, STEPHEN FRANCIS, PARSONS
 MILLIGAN, DONALD B, KANSAS CITY
 MILLS, CHARLES D, WICHITA
 MILLS, KIRK C, KANSAS CITY, MO

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 WIMOSD, JOSE J. DODGE CITY
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 WINNICK, CHARLES V. JUNCTION CITY
 WINNS, GAROLD O. WICHITA
 WIRZA, MEDO, WICHITA
 WISKE, STEPHANIE A. KANSAS CITY
 WISKEW, DON B. W. SHAWNEE MISSION
 MITCHELL, ALEX C. LAWRENCE
 MITCHELL, DEANNA SUE, KANSAS CITY
 MITCHELL, GALEN W. KANSAS CITY
 MITCHELL, JOHN C. SALINA
 MITCHELL, ROBERT H. GARDEN CITY
 WITRA, SUOMEER, GOODLAND
 WITTS, ERNEST W. BONNER SPRINGS
 WOODRELL, CAROL A. LAWRENCE
 WOODLIN, HERBERT C. TOPEKA
 WOEHLER, DONALD O. KANSAS CITY
 WOFFAT, ROBERT E. SHAWNEE MISSION
 WOHLER, JACK M. ABILENE
 WONCKTON, LAURANCE A. LAWRENCE
 MONTGOMERY, LLOYD DAN, HALSTEAD
 MONTGOMERY, THOMAS ALLEN, SABETHA
 MONTGOMERYSHORT, RUTH G. HALSTEAD
 MOORE, DENNIS F. WICHITA
 MOORE, HUGH C. TOPEKA
 MOORE, JAMES E. CONCORDIA
 MOORE, JOHN B. KANSAS CITY
 MOORE, ROBERT, HOISINGTON
 MOORE, ROBERT F. CANEY
 MOORE, WAYNE V. KANSAS CITY
 MOORHEAD JR, F. ALLEN, NEODESHA
 MORGAN, JAMES I. WICHITA
 MORGAN, JOHN L. EMPORIA
 MORGAN, NOVA L. HAYSVILLE
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 MORGAN II, DAVID LLOYD, OLATHE
 MORGAN III, LOUIS S. WICHITA
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 MORONEY, JEAN M. SHAWNEE MISSION
 MORRIS, MERLE D. TOPEKA
 MORRISON, IRA R. ATCHISON
 MORRISON, MICHAEL R. TOPEKA
 MORRISON, RICHARD L. WICHITA
 MORROW, THOMAS F. WICHITA
 MORROW JR, J. TARTON, TOPEKA
 MORTON, JOHN E. HALSTEAD
 MORTON, ROBERT A. ARKANSAS CITY
 MOSELEY, JACK E. WICHITA
 MOSER, ERNEST C. HOLTON
 MOSER, ROY H. HOLTON
 MOSIER, MICHAEL L. MANHATTAN
 MOSIER, STANLEY JAY, WICHITA
 MOSIER, STEVEN J. MANHATTAN
 MOWERY, WILLIAM E. SALINA
 MOWRY, GERALD L. MANHATTAN
 MOYER, HERMAN J. DERBY
 MUEHLBERGER, JAMES J. SHAWNEE MISSION
 MUELLER, ARNOLD V. TOPEKA
 MUELLER, J. KENT, SHAWNEE MISSION
 MUELLER, MICHAEL A. KANSAS CITY
 MUELLER, VERNETTE A. WICHITA
 MUETH, JOAN D. WICHITA
 MULL, JOHN C. HUTCHINSON
 MULLARKY, MATTHEW, BROOKLINE, MA
 MULLEN SR, CLIFFORD J. KANSAS CITY
 MULLER, SAMUEL B. PITTSBURG
 MULLINIK, JANICE M. WICHITA
 MUNOEN, FRANK A. SHAWNEE MISSION
 MURFITT, MALCOLM C. LINDSBURG
 MURPHY, BARRY L. WICHITA
 MURPHY, QUANE A. WICHITA
 MURPHY, JAY W. SHAWNEE MISSION
 MURPHY, MAUREEN E. KANSAS CITY
 MURPHY, PATRICK L. WICHITA
 MURPHY, PAUL M. WICHITA
 MURPHY, TIMOTHY P. SHAWNEE MISSION
 MURPHY, WILLIAM R. KANSAS CITY
 MURPHY, WILLIAM R. C. WICHITA
 MURRAY, KENT B. WICHITA
 MURRAY, W. LEE, SHAWNEE MISSION
 MYERS, JO ANN, TOPEKA
 MYERS, W. EUGENE, IDLA
 MYERS JR, EARL B. INDEPENDENCE
 MYRICK, MICKY, HAYS
 MYRICK, STEPHEN W. LAWRENCE

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NABOURS, RICHARD D. TOPEKA
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 NADEK, DADKAM, KANSAS CITY
 NALDOZA JR, FAUSTINO N. WELLINGTON
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 NOBLE, MARK J. KANSAS CITY
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 NORTH, DORIS G. WICHITA
 NORTH, VICTOR, WICHITA
 NORTON, ROBERT K. WICHITA
 NOSTI, JUAN C. SHAWNEE MISSION
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 NOWLIN, NANCY S. WICHITA
 NUILA, RICHARD F. WICHITA
 NULL, WILLIAM G. SALINA
 NUNEMAKER, MARION E. HUTCHINSON
 NUNEZ, JULIAN, SHAWNEE MISSION
 NYBERG, FREDRIK F. TOWANDA
 NYE, C. ERIK, SHAWNEE MISSION

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 O'NEAL, JON T. WICHITA
 O'NEIL, ROBERT H. TOPEKA
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 O'TOOLE, JAMES K. NORMAN, OK
 OBANDO, GUILLERMO, SALINA
 OBOURN, ROBERT L. TOPEKA
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 OENHEIMER, BURTRAM J. WICHITA
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 OELSCHLAGER, RONALD O. LAWRENCE
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 OHMART, RICHARD V. OAKLEY
 OKTAWIEC, DANUTA, SHAWNEE MISSION
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 OYLER, JONATHAN M. OLATHE

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PACE, JOHN D. PARSONS
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 PAGE, RUTH, WICHITA
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 PAI, VARADARAJ S. PARSONS
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 PATTERSON, JOHN R. SHAWNEE MISSION
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 PAY, NORMAN T. WICHITA
 PAYNE, J. RALPH, KANSAS CITY, MO
 PAYNE, ROBERT R. TOPEKA
 PAZELL, JOHN A. KANSAS CITY
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 PELLETIER JR, LAWRENCE L. WICHITA
 PENCE, CHARLES O. WICHITA
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 PENNER, TIMOTHY M. KANSAS CITY
 PENNINGTON, KATHERINE, WICHITA
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 PEREIRA, WILLY G. ARKANSAS CITY
 PERICO, CARLOS J. KANSAS CITY, MO
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 PERKINS, JACK L. HUTCHINSON
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 PETERSEN, A. GENE, SHAWNEE MISSION
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 PETERSON, JACK T. MANHATTAN
 PETERSON, JAMES E. SALINA
 PETERSON, ROBERT L. TOPEKA
 PETERSON, VERNON J. TOPEKA
 PETERSON JR, EVAN A. WATHENA
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 PFUETZE, BRUCE L. SHAWNEE MISSION
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 PFUETZE, ROBERT E. TOPEKA
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 PHILGREEN, DONALD E. OTTAWA
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 POLLOCK, ANTHONY G. A. WICHITA
 POLLY, RICHARD E. TOPEKA
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 PURINTON, LEW W. WICHITA
 PUTNAM, LYLE B. WICHITA
 PYLE, LUCIEN R. TOPEKA

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 QUIJANO JR, RANDON S. STAFFORD
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 QUINDNES, ELADIO A. TAMPA, FL

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 RANDALL, GORDON R. TOPEKA
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 REDDY, VENUNBABA C. EL DORADO
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 REED, JAMES S. LAWRENCE
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 *REED, WILLIAM RANDALL, WICHITA
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 REMOND, HUBERTO M. ALAIN
 REPLDGL, CHARLES B. GREAT BEND
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 RHODES, LOWELL W. WICHITA
 RICCI, ROBERT LAWLOR, TOPEKA
 RICE, BERNARD F. SHAWNEE MISSION
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 RICH, ELDON S. NEWTON
 RICH, GARY L. KANSAS CITY
 RICH, JOSEPH E. TOPEKA
 RICHARDS, DALLAS LEE, HAYS
 RICHARDS, DENNIS D. CLAY CENTER
 RICHARDSON, CATHERINE A. KANSAS CITY
 RICHARDSON, J. M. TOPEKA
 RICHARDSON, JAY L. KANSAS CITY
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 RICHTER, DON G. SHAWNEE MISSION
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 RIDER, JAMES W. ATCHISON
 RIEDERER, ROBERT E. WICHITA
 RIEGER, ERNEST H. WICHITA
 RIEPE, ROGER E. WICHITA
 RIESENWY, BRANDON D. SHAWNEE MISSION
 RIFFEL, LAWRENCE D. SHAWNEE MISSION
 RIGGS, PAUL A. WICHITA
 RIGGS, SANDRA L. KANSAS CITY
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 RILEY, RAY B. KANSAS CITY
 RINDY, PHILLIP L. FREDDONIA
 RIORDAN, HUGH D. WICHITA
 RIORDAN, TERRANCE, LAWRENCE
 RISING, JESSE D. KANSAS CITY

RITCHIE, KAREN S. SHAWNEE MISSION
 RIZZA, ROBERT G. HALSTEAD
 ROACH, NEIL E. WICHITA
 ROAN, YEAI, WICHITA
 ROBERTS, DANIEL K. WICHITA
 ROBERTS, RICHARD S. LAWRENCE
 ROBERTS, WARREN E. TOPEKA
 ROBERTS, ROGER W. D.O., WICHITA
 ROBERTSON, EDWARD J. SHAWNEE MISSION
 ROBERTSON, JOSEPH K. WICHITA
 ROBINSON, DAVID B. TOPEKA
 ROBINSON, DAVID W. KANSAS CITY
 ROBINSON, EDGAR L. INDEPENDENCE
 ROBINSON, G. DONALD, WICHITA
 ROBINSON, JOHN D. SHAWNEE MISSION
 ROBINSON, JOHN E. WICHITA
 ROBINSON, RALPH G. KANSAS CITY
 ROBINSON, ROBERT H. WICHITA
 ROBINSON, JAMES T. SHAWNEE MISSION
 ROBL, DAVID A. WICHITA
 ROCHANAYDN, PIRA, ELLIS
 RODERICK, JAMES E. SALINA
 RODRIGUEZ, ALBERTO, KIDWA
 RODRIGUEZ, PAUL L. GARDEN CITY
 RODRIGUEZ-RAWDS, ERNEST R. WICHITA
 RODRIGUEZTODCKER, LILIA, WICHITA
 ROEDER, ROBERT E. TOPEKA
 ROENBACH, JEANINE L. TOPEKA
 ROWALIS, BRIAN E. WICHITA
 ROWE, MICHAEL P. KANSAS CITY
 ROWEISER, REK S. SALINA
 RUMONDO, STEVEN A. OLATHE
 ROK, LEE E. KANSAS CITY
 ROOS, MAUREEN, WICHITA
 RORABAUGH, DONALD C. ABILENE
 ROSE, DONALD L. BELLA VISTA, AR
 ROSE, GRAHAM C. MANHATTAN
 ROSE, SHELBY D. WICHITA
 ROSEN, DAVID, WICHITA
 ROSENBERG, STANTON L. SHAWNEE MISSION
 ROSENBERG, THOMAS F. WICHITA
 ROSENTHAL, STANTON J. KANSAS CITY
 ROSIN, ROBERT L. WICHITA
 ROSS, ALBERT M. SHAWNEE MISSION
 ROSS, DAVID K. ARKANSAS CITY
 ROSS, DENNIS LEE, WICHITA
 ROSS, JACK L. TOPEKA
 ROTERT, LARRY, TOPEKA
 ROTH, ALAN E. KANSAS CITY
 ROTHSTEIN, TERRY B. PARSONS
 ROWLETT, JACK G. PADLA
 RDY, WILLIAM R. TOPEKA
 RUBIN, HERBERT M. SHAWNEE MISSION
 RUBIN JR, BEN, KANSAS CITY
 RUBLE JR, JAMES L. DYERBROOK
 RUEB, ANDREW E. SALINA
 RUHL, CONSTANCE E. KANSAS CITY, WD
 RUHLEN, JAMES L. DLAHE
 RUIZ, CARLOS M. GREAT BEND
 RUMOLD, WERVIN J. SHAWNEE MISSION
 RUNNELS, JOHN B. TOPEKA
 RUPP, RICHARD J. TOPEKA
 RUSSELL, PHILIP W. WICHITA
 RUSSO, LIBBIE J. KANSAS CITY
 RUTH, WILLIAM E. KANSAS CITY
 RUTNGAMLUG, LUECHA, HAYS
 RUZICKA, LAWRENCE J. CONCORDIA
 RYAN, MICHAEL E. SHAWNEE MISSION
 RYAN, MICHAEL J. KANSAS CITY
 RYAN, W. SCOTT, EWPORIA
 RYWER, ROBERT A. SHAWNEE MISSION

S

SABIN JR, GEORGE W. WICHITA
 SACHEN, FREDERICK L. SHAWNEE MISSION
 SACK, JOSEPH W. KANSAS CITY
 SADIQ, SULEWAN, WICHITA
 SAEED, MOHAMMAD, WICHITA
 SAFFD, KARL S. SHAWNEE MISSION
 SALEN, GEORGE A. D.O., KANSAS CITY, WD
 SALMON, JAMES S. KANSAS CITY
 SAWPAT, PRAVIN, TOPEKA
 SAMUEL, CHANDY C. WINFIELD
 SAMUEL, SHANTHI, WINFIELD
 SANCHEZ, ROGELIO, TOPEKA
 SANDERS, J. ALAN, LAWRENCE
 SANDERS, JAMES E. KANSAS CITY, WD
 SANDHU, PAUL S. COFFEYVILLE
 SANDSCDY, GILBERT S. WICHITA
 SARGENT, JOSEPH D. TOPEKA
 SATHYANARAYANA, SARASWATHI, SHAWNEE MISSION
 SAUL, F. WILLIAM, MECHANICSBURG, PA
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 SAWKAR, LAKSHIDAS A. SHAWNEE MISSION
 SAYEED, BASEER A. HAYSVILLE
 SAYLER, JEROME, GREAT BEND
 SAYLOR, EDWARD M. TOPEKA
 SAYLOR, LESLIE L. TOPEKA
 SAYLOR, MARK, TOPEKA
 SAYLOR, STEPHEN, TOPEKA
 SCALES, WILLIAM W. HUTCHINSON
 SCANMAN, W. WIKI, TOPEKA
 SCANLAN, TIDOTHY W. SALINA
 SCHAEFER, JOSEPH PETER, SHAWNEE MISSION
 SCHAEFFER, CLARENCE K. SANTA CRUZ, CA
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 SCHIMKE, R. NEIL, KANSAS CITY
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 SCHLACHTER, ERNEST R. WICHITA
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 SCHLOTTERBACK, WILLIAM E. BELLEVILLE
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 SCHOTLAND, EDWARD S. KANSAS CITY
 SCHRAM, PETER CHARLES, TOPEKA
 SCHREFFER, ROSEMARY, SHAWNEE MISSION
 SCHROEDER, SYDNEY D. LAWRENCE
 SCHROLL, JACK C. HUTCHINSON
 SCHROLL, JOHN T. SHAWNEE MISSION
 SCHUETZ, PERRY N. GREAT BEND
 SCHUKMAN, JAY S. GREAT BEND
 SCHULTZ, JAMES E. COUNCIL GROVE
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 SCHURLE, DALE R. MCPHERSON
 SCHUSTER, MICHAEL R. SHAWNEE MISSION
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 SCHWEGLER, RAYMOND A. LAWRENCE
 SCHWEGLER, RAYMOND A. KANSAS CITY
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 SCOTT, DUANE L. BELLEVILLE
 SCOTT, JEFFREY, KANSAS CITY
 SCOTT, JUDITH C. SHAWNEE MISSION
 SCOTT, STEVE G. KANSAS CITY, MO
 SCOTT, WILLIAM H. WICHITA
 SEAMAN, LAUREN T. OLATHE
 SEATON, ROBERT D. SALINA
 SEBREE, STEVEN G. SALINA
 SEGBRECHT, STEPHEN L. LAWRENCE
 SEGERSON, JOHN A. TOPEKA
 SEGUE, FLOYD RONALD, PITTSBURG
 SEGRAVES, STEVEN D. SHAWNEE MISSION
 SEHOEV, JOAN, TOPEKA
 SEIFERT, EARNEST D. KANSAS CITY
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 SEVIER, SAMUEL M. MUSKOGEE, OK
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 SHAH, MIAN, LARNED
 SHAH, MUKHTAR H. WICHITA
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 SHARMA, ARUN L. PARSONS
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 SHAW, PAMELA K. KANSAS CITY
 SHAW, RICHARD C. WICHITA
 SHEAFOR, DOUGLAS, TOPEKA
 SHEARS, ROBERT N. HUTCHINSON
 SHEEHAN, MAUREEN H. KANSAS CITY
 SHEERN, MARK DOUGLAS, ABILENE
 SHEFFER, KEITH D. OLATHE
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 SHIVEL, DAVID G. GREAT BEND
 SHOFFNER, RICHARD W. WICHITA
 SHOPSTALL, WILLIAM H. SHAWNEE MISSION
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 SHRAIDER, C. ERIC, WICHITA
 SHRAIDER, DOYLE A. WICHITA
 SHULTZ, WILLIAM H. SHAWNEE MISSION

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 SISK, PHILLIP B. TOPEKA
 SIWEK, CHRISTOPHER W. EL DORADO
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 SKIBBA, RICHARD M. WICHITA
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 SKOCH, MICHAEL G. WICHITA
 SLAVIK, MILAN, KANSAS CITY, MO
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 SMITH, ALVIN L. WICHITA
 SMITH, BOYO E. SALINA
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 SMITH, NEWTON C. ARKANSAS CITY
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 SMITH, THOMAS WILLIAM, HUTCHINSON
 SMITH, TIMOTHY W. WICHITA
 SMITH, WILLIAM P. SHAWNEE MISSION
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 SMITH JR., WILLARD J. WICHITA
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 SNYDER, H. MARTIN, WINFIELD
 SNYDER, HOWARD E. WINFIELD
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A	Allergy	NP	Neuropsychiatry
ADL	Adolescent Medicine	NR	Nuclear Radiology
ADM	Administrative Medicine	NS	Neurological Surgery
ADT	Addictionology	OBG	Obstetrics and Gynecology
AM	Aviation Medicine	OM	Occupational Medicine
ANES	Anesthesiology	ON	Oncology
BLB	Blood Bank	OPH	Ophthalmology
CD	Cardiovascular Disease	ORS	Orthopedic Surgery
CDS	Cardiovascular Surgery	OS	Other Specialties
CDTS	Cardiovascular & Thoracic Surgery	OST	Osteopathy
CHN	Child Neurology	OTO	Otorhinolaryngology
CHP	Child Psychiatry	P	Psychiatry
CP	Clinical Pharmacology	PATH	Pathology
D	Dermatology	PD	Pediatrics
DR	Diagnostic Roentgenology	PDA	Pediatric Allergy
EENT	Eye, Ear, Nose and Throat	PDC	Pediatric Cardiology
EM	Emergency Medicine	PDE	Pediatric Endocrinology
END	Endocrinology	PDN	Pediatric Neurology
ENT	Ear, Nose, Throat	PDO	Pediatric Ophthalmology
ES	Endoscopy Surgery	PDR	Pediatric Radiology
FM	Family Medicine	PDS	Pediatric Surgery
FP	Family Practice	PGER	Psychogerontology
GE	Gastroenterology	PH	Public Health
GEN	Genetics	PM	Physical Medicine & Rehabilitation
GER	Geriatrics	PNP	Pediatric Nephrology
GP	General Practice	PS	Plastic Surgery
GPM	General Preventive Medicine	PUD	Pulmonary Disease
GPVS	General & Peripheral Vascular Surgery	PYM	Psychosomatic Medicine
GS	General Surgery	R	Radiology
GYN	Gynecology	RES	Resident
HEM	Hematology	RHU	Rheumatology
ID	Infectious Diseases	RM	Rehabilitative Medicine
IE	Insurance Examination	RN	Radiology & Neurology
IM	Internal Medicine	RO	Radiology/Oncology
MDST	Medical Student	RT	Radiation Therapy
MFM	Maternal Fetal Medicine	SM	Sports Medicine
MO	Medical Oncology	SON	Surgical Oncology
N	Neurology	TR	Therapeutic Radiology
NEP	Nephrology	TS	Thoracic Surgery
NM	Nuclear Medicine	U	Urology
NEO	Neonatology	00	Retired

Physician Distribution by Cities

EXPLANATION OF CODES USED IN THIS SECTION

Line 1: Doe, John R., 1234 Oak St., 67052
 (Name) (Street Address) (Zip Code)

Line 2: (654-2222)
 (Telephone Number)

Line 3: 33 M 1902 58 FP
 (Year of Birth) (Sex) (Medical School) (Year of Graduation) (Specialty)

Telephone area code follows city name. * Probationary Members

ABILENE—913 (Dickinson County Society)

BERKLEY, DON H. 1111 N BRADY, 67410
 263-4131
 35 M 1902 61 FP
 BIGGS, DENNIS, 1405 N CEDAR, 67410
 263-7190
 48 M 1902 74 FP
 CHAFFEE, DEAN C. RR 1, 67410
 -
 11 M 1902 44 OD
 COLEMAN, GARY, 1405 N CEDAR, 67410
 263-7190
 46 M 1902 72 FP
 MOHLER, JACK M. 515 NE TENTH, 67410
 263-1419
 32 M 1902 61 PM
 NARCISO, VICENTE O. 515 NE 10TH ST, 67410
 263-2253
 45 M 74810 68 GS
 RORABAUGH, DONALD C. PROFESSIONAL BLDG, 67410
 263-4131
 33 M 1902 58 FP
 SCHWARTING, J STEVE, 1405 N CEDAR, 67410
 263-7190
 46 M 3401 72 FP
 SHEERN, MARK DOUGLAS, 1111 N BRADY, 67410
 263-4131
 51 M 1902 76 FP
 SONGER, HERBERT L. 811 SPRUCE WAY, 67410
 -
 12 M 1902 38 OD

ALMA—913 (Pottawatomie County Society)

MCKNIGHT, ELLIS B. 66401
 765-3317
 03 M 1902 33 FP

ALTAMONT—316 (Labette County Society)

JACKSON, VICTOR L. BOX 467, 67330
 784-5318
 20 M 2105 50 FP

ANDALE—316 (Sedgwick County Society)

GDOOWIN, MARY K. ANDALE CLINIC, 67001
 267-0865
 53 F 1902 77 FP
 STECH, JOSEPH M. ANDALE CLINIC, 67001
 445-2152
 27 M 3006 56 FP

ANDOVER—316 (Sedgwick County Society)

KLINGMAN, DIANE D. PO BOX 426, 67002
 733-1331
 53 F 1902 79 FP

ANTHONY—316 (Tri-County Society)

ANTRIM, PHILIP JENIFER, 1101 E SPRING, 67003
 842-5144
 15 M 1902 42 FP

ARKANSAS CITY—316 (Cowley County Society)

ALVAREZ, NORBERTO, 112 E CENTRAL, 67005
 442-4850
 27 M 27501 53 FP
 AUCAR, ALFREDO, BOX 1105, 67005
 442-1710
 23 M 27501 53 OTD
 BAKER, ROBERT R. O.O., 2508 EDGE MONT BOX 714, 67005
 442-8924
 50 M 2878 78 FP
 CAMPBELL, GARLAND L. 114 W WALNUT, 67005
 442-1350
 13 M 1902 40 U
 GREEN, LAWRENCE C. PO BOX 173, 67005
 442-4850
 43 M 3901 69 FP
 HINSHAW, EDGAR O. RT #3, 67005
 442-1273
 15 M 1902 51 R
 LEIKER, JOSEPH, 112 E CENTRAL, 67005
 442-7900
 48 M 1902 73 IM
 MARVEL, JAMES EBBERT, 2508 EDGE MONT, 67005
 442-0222
 43 M 3901 72 JRS
 MORTON, ROBERT A. AC OFFICE BLDG, 67005
 442-0370
 51 M 1902 73 IM
 DLO, JERRY L. PO BOX 1148, 67005
 442-2100
 49 M 1902 74 FP
 PEREIRA, WILLY G. 2508 EDGE MONT DR, 67005
 442-8540
 39 M 73701 67 IM
 ROSS, DAVID K. PO BOX 1148, 67005
 442-2100
 48 M 1902 75 FP
 SCHMEIDLER, DAVID ALLEN, 510 W RADIO LANE, 67005
 442-2100
 54 M 1902 79 FP
 SINGH, GIRVAR, 2508 EDGE MONT DR, 67005
 442-4300
 40 M 49555 64 OPH
 SMITH, BRUCE G. 115 E RADIO LANE, 67005
 442-5600
 20 M 1902 44 IM
 SMITH, NEWTON C. PO BOX 1148, 67005
 442-2100
 21 M 3901 45 FP
 YOACHIM, ROBERT W. 510 W RADIO LANE, 67005
 442-2100
 52 M 3005 78 FP

ARMA—316 (Crawford County Society)

RIGLER, WILSON F. 511 WASHINGTON, 66712
 347-8619
 42 M 1803 69 FP

ASHLAND — 316
(Iroquois County Society)

DION, MARK W., 529 W SEVENTH, 67831
-
51 M 702 77 GP

ATCHISON—913
(Atchison County Society)

BOSSE, FRANK K., 1301 RIVERVIEW DRIVE, 66002

09 M 2802 33 DO
BRADY, CHARLES S., PD BDX 245, 66002
367-1232
11 M 3006 38 GS
BRIBACH, EUGENE J., 125 1/2 N 5TH, 66002
367-0225
83 M 2802 05 DPH
BRDWN, ROBERT O., 1400 NORTH 2ND, 66002
367-1922
14 M 1902 44 FP
BURKE, JOSEPH V., 1301 N 3RD, 66002
367-5496
35 M 3006 66 GS
DUNLAP, MICHAEL D., 1225 N SECOND, 66002
367-0362
53 M 1902 79 FP
EPLER, JOHN R., 1225 N SECOND, 66002
367-0362
53 M 1902 78 FP
FAST, ROBERT E., 1225 N 2ND, 66002
367-0362
48 M 1902 74 DBG
FAST, W SPENCER, RAMSAY MED CLINIC, 66002
367-0362
11 M 3006 39 FP
GROWNEY, JOHN T., 801 ATCHISON, 66002
367-5020
37 M 3006 63 FP
HART, LAWRENCE E., 1412 N 2ND, 66002
367-5054
32 M 1902 64 FP
MESROBIAN, GEORGE D., 1225 N SECOND, 66002
367-1114
48 M 60501 74 GS
MORRISON, IRA R., 825 N 10TH, 66002
-
07 M 1611 36 1M
RIDER, JAMES W., 1225 N 2ND ST, 66002
367-0362
47 M 2803 73 FP
TIVORSK, ARKDM, 1716 COUNTRY LANE, 66002
367-2131
40 M 89101 68 R
VESOM, PITT, 1301 N SECOND, 66002
367-3100
49 M 89104 74 CD
WALLACE JR, WAYNE D., 1301 N THIRD, 66002
367-7300
36 M 2803 65 FP
WULFF, EDWIN T., 923 N FIFTH, 66002
367-5033
07 M 2834 36 FP
YOUNG, CHARLES H., 1301 N 3RD, 66002
367-4053
23 M 1902 53 FP

ATTICA—316
(Tri-County Society)

STONE, GRANT C., 500 N HARPER, 67009
254-7219
09 M 5605 35 FP

ATWOOD—316
(Northwest Kansas Society)

DILL, RODNEY, PJ BDX 67, 67730
626-3229
41 M 74811 77 GP
ERBACHER, GEORGE E., D.O., PD BDX 203, 67730
626-3238
51 M FP

AUGUSTA—316
(Butler-Greenwood Society)

ANDERSON, DALE W., 120 W JOSEPHINE, 67010
775-5432
30 M 1902 55 FP
BARBER, JAMES L., 120 W JOSEPHINE, 67010
775-5432
31 M 1902 57 FP
TUONG, TRAN MANH, 120 W JOSEPHINE, 67010
775-5432
39 M 94101 65 FP

BAXTER SPRINGS—316
(Cherokee County Society)

ALQUIST, VERYL D., 21ST & FAIRVIEW, 66713
623-4942
17 M 1902 42 GS
CHUBB, RICHARD M., 445 EAST 10TH, 66713
855-2144
29 M 1606 54 FP

BELLEVILLE—913
(Republic County Society)

BEIDERWELL, PAUL L., 2703 M ST, 66935
-
08 M 3901 38 FP
DOUBEK, HERBERT D., PD BDX 250, 66935
527-2237
28 M 1902 56 FP
SCHLOTTERBACK, WILLIAM E., 1202 L, 66935
378-3511
31 M 1902 61 FP
SCOTT, DUANE L., BELLEVILLE CLINIC, 66935
527-2217
34 M 1902 60 FP
WARD, JAMES A., 1206 18TH ST, 66935
527-2217
34 M 1902 58 FP

BELOIT—913
(Mitchell County Society)

DOBRTZ, ROBERT A., 310 W 8TH, 67420
738-2246
24 M 1902 52 FP
DRAKE, DOUGLAS J., MEDICAL CENTER, 67420
738-2246
43 M 1902 71 FP
KLEND JR, MARTIN B., BELDIT MED CENTER, 67420
738-2246
38 M 1643 63 GS
LDNEY, JOHN M., 310 WEST 8TH, 67420
738-2246
50 M 1902 74 1M
WELTNER, ROGER P., 112 W MAIN, 67420
738-2574
18 M 1902 44 U

BLUE RAPIDS—913
(Northeast Kansas Society)

LAWLESS, HAROLD L., 607 LINCOLN, 66411
226-7202
29 M 702 54 FP

BONNER SPRINGS—913
(Wyandotte County Society)

DETAR NEWBERT, LE ANNE, PD BOX 41, 66012
-
51 F 1902 87
FUNK, EDWARD D., 216 E SECOND, 66012
441-2646
04 M 1902 41 ANES
MAY, KENNETH L., MAC GRANTWOOD RD, 66012
422-2020
20 M 1902 51 FP

MITTS, ERNEST W. 122 N NETTLETON.66012
422-2020
22 M 1902 51 FP
WAGGONER, FRANKLIN E. 122 N NETTLETON.66012
422-2020
26 M 1902 61 FP

BUHLER—316
(Reno County Society)

FRIESEN, ORLANDO J. 107 W 2ND.67522
543-2330
27 M 1902 56 FP

BURLINGAME—913
(Shawnee County Society)

GOERING, EMIL L. 130 W SANTA FE.66413
654-2300
27 M 1902 57 14

CALDWELL—316
(Tri-County Society)

KINNAN, L F. 523 S MARKET.67022
845-6422
18 M 3901 42 FP

CANEY—316
(Southeast Kansas Society)

MOORE, ROBERT F. 4TH & MCGEE.67333
879-2135
28 M 1902 56 FP

CEDAR VALE—316
(Southeast Kansas Society)

COHNBERG, ROSELLEN E. PO BOX 398.67024
758-2266
22 F 2802 47 FP

CHANUTE—316
(Southeast Kansas Society)

ABBUEHL, DON R. 505 SOUTH PLUMMER.66720
431-2500
18 M 1902 44 GS
ASHLEY, SAMUEL G. 505 SOUTH PLUMMER.66720
431-2500
16 M 1902 43 FP
BAKER, HENRY K. 1220 W FOURTH.66720
431-1600
08 M 1606 35 GS
BURKMAN, REUBEN J. 1501 W 7TH.66720
431-9310
28 M 1902 54 FP
DICK JR, HENRY J. 1501 W 7TH.66720
431-9310
27 M 1902 58 FP
GAREY, WILLIAM JOHN, PO BOX 321 RT 2.66720
431-4000
44 M 6501 70 OR
GEHRT, EARL B. 505 SO PLUMMER.66720
431-2500
32 M 1902 62 FP
HASKINS, ROBERT J. 505 S PLUMMER.66720
431-2500
46 M 1902 74 FP
KIMM, ALBERT A. 505 S PLUMMER.66720
431-2500
27 M 1902 55 FP
MABEY, PAMELA S. 505 S PLUMMER.66720
431-2500
54 F 1902 79 IM
MIM, ALEXANDER. 1002 WEST 4TH.66720
473-2227
22 M 24209 47 ANES
PARHAM, VERDON W. 505 S PLUMMER.66720
431-2500
47 M 1902 73 FP

VAN HOUDEN, CHARLES E. 505 S PLUMMER.66720
431-2500
52 M 1902 77 GS

CHAPMAN—913
(Dickinson County Society)

SVOBODA, CHARLES R. 413 N MARSHALL.67431
922-6400
18 M 1902 46 FP

CHETOPA—316
(Labette County Society)

PEFFLY, ELMER O. 327 MAPLE.67336
236-7188
22 M 3901 53 FP

CLAY CENTER—913
(Clay County Society)

ANDERSON, SEVERT A. 1749 BERGLUND DR.67432
-
07 M 1902 36 00
BOGNER, PAUL F. 1318 SUNRISE CIRCLE.67432
632-2185
52 M 1902 77 GS
BRYANT, ROONEY K. 709 LIBERTY.67432
632-2181
54 M 515 79 FP
CARLETON, RICHARD C. 709 LIBERTY.67432
632-5603
31 M 3005 61 FP
DALUM, PETER JOSEPH. 709 LIBERTY SUITE C.67432
632-2181
45 M 2803 76 FP
GOOD, MICHAEL W. 709 LIBERTY.67432
632-2181
53 M 1902 78 FP
O'DONNELL, RICHARD H. PO BOX 806.67432
632-3101
16 M 1902 41 GS
RICHARDS, DENNIS O. 115 S 6TH.67432
632-5621
34 M 1902 60 FP

CLYDE—913
(Cloud County Society)

COULTER, THAYNE A. O.D.. 306 N HIGH.66938
-
12 M 2878 37 00
FREEBORN JR. WARREN S. .66938
446-2221
26 M 1720 51 FP

COFFEYVILLE—316
(Southeast Kansas Society)

BANKS, GILBERT, PO BOX 257.67337
251-7500
49 M 2401 75 IM
CAMPBELL, WILLIAM H. 1411 W 4TH.67337
251-3235
39 M 1902 65 OPH
COYLE, JOHN F. PO BOX 487.67337
251-2400
21 M 1902 44 FP
DICKINSON, CHARLES R. 108 W 7TH.67337
251-1340
20 M 1606 44 GS
DIXON, RAYMOND W. 1411 W 4TH.67337
251-1090
46 M 4706 71 GS
EDRZO, M LUZ LUNA, PO BOX 497.67337
251-1200
44 F 74801 68 PATH
GIBBS, EUGENE, PO BOX 716.67337
251-7260
- M 64914 68 FP
HA, SANG W. 504 WILSHIRE.67337
251-7750
35 M 58309 60 OBG

HAN, CHAN S. 908 SIGGINS.67337
 251-1560
 35 M 58306 61 PD
 HOWERTER JR. BERNARD E. PO BOX 659.67337
 251-4790
 43 M 1803 68 U
 JIRICKO, WILDS. 2510 W EIGHTH.67337
 251-1200
 37 M 28601 61 ANES
 NICHOLS, RICHARD. 1411 W FOURTH.67337
 251-6540
 M 1803 73 ORS
 READ, WILLIAM T. 1411 WEST 4TH.67337
 251-1120
 16 M 2802 40 FP
 SANDHU, PAUL S. PO BOX 257.67337
 251-2450
 42 M 49508 65 GS
 UY, WILSON D. COFFEYVILLE MEM HOSPITAL.67337
 251-1200
 42 M 74801 67 PATH
 VAKAS, JOHN L. 1508 W 4TH.67337
 251-3443
 38 M 1902 64 IM
 WHITE, DONALD C. PO BOX 1449.67337
 251-1200
 35 M 3515 65 R

COLBY—913
(Northwest Kansas Society)

DAHL, ASHER W. COLBY CLINIC.67701
 462-3333
 28 M 1902 58 FP
 HASSETT, GERARD R. 1875 HARVEY.67701
 -
 24 M 3006 50 R
 HILOYARD II. VICTOR H. BOX 28.67701
 462-3332
 47 M 702 73 FP
 JACOBSEN, DWIGHT SKINNER. COLBY MED & SURG CTR.67701
 462-3333
 31 M 3545 60 GS
 KOSTE, REX J. COLBY MED & SURGICAL CTR.67701
 462-3332
 53 M
 REGIER, LADDNA M. COLBY CLINIC.67701
 462-3332
 47 F 1902 73 FP
 SMITH JR. FLOYD L. DRAWER K.67701
 462-3333
 20 M 1902 44 FP
 WINGER, RAYMOND E. COLBY MED & SURGICAL CTR.67701
 462-7971
 51 M

COLDWATER—316
(Iroquois County Society)

GDERING, DONALD D. BOX 748.67029
 582-2136
 31 M 1902 56 FP

COLUMBUS—316
(Cherokee County Society)

ANDREASEN, RAYMOND L. 219 S KANSAS.66723
 429-3744
 47 M 1720 74 FP
 BELCHER, GEORGE D. BOX 309.66725
 429-2557
 34 M 1902 60 FP
 JONES, FORREST H. 219 S KANSAS.66725
 429-3744
 25 M 1902 54 FP
 PASIMID, ROGER S. R2 BOX 259.66725
 429-1977
 38 M 74801 62 GS

COLWICH—316
(Sedgewick County Society)

LIES, BARTHEL N. 309 S THIRD.67030
 -
 11 M 2802 37 OD

CONCORDIA—913
(Cloud County Society)

BRAY, AVIS PAGE. 1010 3RD AVE.66901
 243-1560
 17 F 702 54 FP
 BUTT, MUHAMMAD. 1010 THIRD AVE.66901
 243-1560
 46 M 70401 69 GS
 CORNELL, EARL G. 1010 THIRD AVE.66901
 243-1560
 54 M 1902 79 FP
 FOWLER, WAYNE L. 1010 THIRD.66901
 243-1560
 23 M 1720 47 IM
 GELVIN, E RAYMOND. 835 WEST 9TH.66901
 243-1560
 03 M 3005 27 GS
 GRANT, MICHAEL D. 1010 THIRD.66901
 243-1560
 46 M 1902 79 FP
 HOFER, DEWAYNE D. COUNTRY CLUB DR.66901
 243-1263
 36 M 1606 62 R
 KOSAR, CLARENCE D. BOX 362.66901
 -
 98 M 1902 26 OD
 LAWTON, MARVIN K. 1010 THIRD.66901
 243-1560
 31 M 3005 58 GS
 LLOYD, JAMES W. 910 WEST 11TH.66901
 243-7011
 44 M 1902 73 FP
 MOORE, JAMES E. PO BOX 305.66901
 243-7636
 48 M 1902 74 P
 NELSON, PAUL L. 1010 THIRD AVE.66901
 243-1560
 27 M 1902 55 PD
 NEWMAN, CARL T. PO BOX 587.66901
 243-2511
 49 M 64914 76 U
 OWENSBY, L C. 222 W SIXTH.66901
 243-3386
 21 M 2802 44 DPH
 RUZICKA, LAWRENCE J. 1115 HILLSIDE DR.66901
 243-1560
 13 M 3005 40 ANES
 SAUNDERS, MICHAEL E. 10 LDST CREEK LANE.66901
 243-1560
 50 M 1902 73 IM
 STRYKER JR. HENRY B. 717 FIRST AVE.66901
 -
 19 M 3501 44 OD
 THORNTON JR. FOXHALL P. 723 W 7TH ST.66901
 243-1560
 25 M 5101 50 IM
 WOLF, PATRICK G. 1413 COUNTRY CLUB DR.66901
 243-1560
 52 M 1902 77 IM

COTTONWOOD FALLS—316
(Flint Hills Society)

BROWNING, JIMMIE L. BOX 486.66845
 273-6753
 50 M 1902 78 FP
 MCKEE, LEO F. .66845
 273-6681
 16 M 1902 39 FP

COUNCIL GROVE—316
(Flint Hills Society)

ALLRED, CHARLES T. 221 HOCKADAY.66846
 767-5126
 53 M 1902 78 FP
 BARKER, ROYAL A. 221 HOCKADAY.66846
 767-5126
 21 M 1902 53 FP
 BLACKBURN, ROBERT W. 221 HOCKADAY.66846
 767-5126
 22 M 1902 49 FP
 SCHULTZ, JAMES E. 221 HOCKADAY.66846
 767-5126
 26 M 1902 56 FP

CUNNINGHAM—316
(*Ninnescah Society*)

ALLBRITTEN JR., FRANK F. PO BOX 177, 67035
14 M 4101 38 00

DE SOTO—913
(*Johnson County Society*)

POMMERENKE, FORREST A. 206 E SECOND, 66018
585-1177
48 M 1902 75 FP

DENTON—913
(*Northeast Kansas Society*)

YODER, EMERSON O. , 66017
359-6531
14 M 1902 49 FP

DERBY—316
(*Sedgwick County Society*)

MCKERRACHER, ROBERT D. 400 A NORTH BALTIMORE, 67037
688-1779
27 M 3901 55 FP
MILLER, LAWRENCE H. 606 MULBERRY, 67037
788-3741
40 M 1001 67 FP
MILLER, ROGER M. 5901 S GREENWICH RD. 67037
268-0830
37 M 4102 63 8L8
MOYER, HERMAN J. 200 S BALTIMORE, 67037
788-1484
24 M 3901 55 FP
VINZANT, MARK N. 1410 N WOODLAWN, 67037
681-3092
45 M 64914 75 FP

DIGHTON—316
(*Southwest Kansas Society*)

VON LEONROD JR., GEORGE. PO BOX 146, 67839
397-5314
16 M 1902 43 FP

DODGE CITY—316
(*Ford County Society*)

AMAWI, MOHAMMAD S. DODGE CITY MED CENTER, 67801
225-1371
46 M 87501 71 GS
AVILA, OSCAR A. DODGE CITY MED CENTER, 67801
225-1371
41 M 17603 69 IM
AYUTHIA, ISSARA I. 2004 FREDERICK DR, 67801
40 M 89101 66 PATH
BAUM, ARNOLO H. 2214 TRIM AVE, 67801
225-1371
16 M 2101 44 OBG
BOLES, R OALE, DODGE CITY MED CENTER, 67801
225-1371
23 M 3901 53 PO
BROWNRIGG, RICHARD L. 108 ROSS BLVD, 67801
225-0075
35 M 1902 61 U
BUSCH, ANTHONY B. SOUTHWEST CLINIC, 67801
05 M 2002 31 FP
CHOTIMONGKOL, ANUPONG, DODGE CITY MED CENTER, 67801
225-1371
43 M 89102 69 OBG
CONARD, CLAIR C. DODGE CITY MED CENTER, 67801
225-1371
27 M 1902 55 IM
GARCIA, GUILLERMO O. 1206 FRONTVIEW, 67801
225-7710
43 M 23101 68 ORS

GREENBERG, GEORGE E. 1904 BURR PARKWAY, 67801
276-8241
42 M 401 68 R
JAMBOR, CHRISTOPHER N. DODGE CITY MED CENTER, 67801
225-1371
51 M 1902 78 PD
JOHNSON, HOWELL O. DODGE CITY MED CENTER, 67801
225-1371
45 M 1902 71 IM
MCCOY, RONALD. SOUTHWEST CLINIC, 67801
227-3141
19 M 3901 47 FP
MCELHINNEY, CHARLES F. DODGE CITY MED CENTER, 67801
225-1371
36 M 1902 62 GS
MCGINNIS, MICHAEL J. SOUTHWEST CLINIC, 67801
227-3141
41 M 5501 69 GS
MINDOS, JOSE J. SOUTHWEST CLINIC, 67801
227-3141
37 M 4201 61 OBG
NIXON, JAMES E. 2008 BURR PARKWAY, 67801
225-1033
40 M 4812 72 OR
OHMAN, RICHARD J. 1810 1/2 FAIRWAY DR, 67801
15 M 2407 41 00
PIZARRO, FE E. 806 SECOND, 67801
227-3141
44 F 74810 68 PO
REDDY, SATTI S. 108 RUSS BLVD, 67801
225-0075
35 M 49504 66 U
SCHWARTZ, EUGENE W. 1ST NATL BANK BLDG, 67801
225-4261
24 M 1902 50 OPH
STOCKWELL, MORGAN U. 1304 GRAY, 67801
225-1371
24 M 1902 55 IM
TREKELL, WILLIAM V. SOUTHWEST CLINIC, 67801
227-3141
25 M 1902 52 ORS
TROTTER, ROGER COURTNEY. 1111 SIXTH AVE, 67801
225-6120
47 M 1902 74 FP
VIERTHALER, CARL A. DODGE CITY MED CENTER, 67801
225-1371
53 M 1902 78 IM
ZACHARIAS, CARL KURT. DODGE CITY MED CENTER, 67801
225-1371
21 M 40707 47 ORS

EL DORADO—316
(*Butler-Greenwood Society*)

BRIAN, ROBERT M. 123 N ATCHISON, 67042
321-1230
02 M 1606 30 FP
DELLETT, KENNETH B. 3RD & VINE, 67042
321-1910
30 M 1902 55 OPH
GIROD, CHARLES I. 123 N ATCHISON ST #103, 67042
321-4981
11 M 4706 44 GS
HAFFNER, WILLIAM N. 123 N ATCHISON, 67042
321-5630
35 M 1902 61 GS
JACOB, KANNAMPALLY L. 123 N ATCHISON, 67042
321-0056
31 M 49537 59 U
KASSEBAUM, GLEN E. 123 N ATCHISON, 67042
98 M 1606 23 GS
LEE, YONG U. 123 N ATCHISON, 67042
321-5630
35 M 58310 60 GS
OLSEN, PHILLIP S. 123 N ATCHISON, 67042
321-2100
46 M 1902 73 IM
OVERHOLSER, NORMAN H. 119 N JONES, 67042
321-2010
16 M 1902 41 FP
PROCTOR, ROBERT W. 123 N JONES, 67042
321-2010
38 M 1902 63 FP
REDDY, VENUMBAKA C. ROOM 302, 67042
321-3300
46 M 49511 70 IM
SHIELOS JR, JAMES M. 119 N JONES, 67042
321-2010
18 M 4802 42 FP

SIWEK, CHRISTOPHER W. 123 N ATCHISON.67042
321-5211
48 M 75911 71 ORS
WHITE II, BENJAMIN E. 119 N JONES.67042
321-2010
27 M 1902 54 FP

ELKHART—316
(Seward County Society)

CABRERA, ALBERTO, BOX 997.67950
697-2155
30 M 74801 55 GS
CABRERA, NATIVIDAD R. 411 SUNSET DR.67950
697-2153
31 F 74801 56 OBG
WAY, BELINDO D. 411 SUNSET BOX 460.67950
697-2175
42 M 74811 66 IM
WAY, OLIVIA N. BOX 460 - 411 SUNSET.67950
697-2175
43 F 74811 68 P
PERIOD, DOMINADOR T. BOX 997.67950
697-2155
44 M 74801 68 GS

ELLINWOOD—316
(Barton County Society)

LAW, FINDLEY, MEDICAL ARTS BLDG.67526
564-2170
22 M 1902 51 FP
SHAPIRO, MARTIN, MEDICAL ARTS BLDG.67526
564-2170
54 M 74809 81 GP

ELLIS—913
(Central Kansas Society)

ROCHANAYON, PIRA, 1204 WASHINGTON.67637
726-4956
43 M 89101 69 FP
SURFACE, GARDNER A. 101 WEST 13TH.67637
726-4454
02 M 1902 29 OO

ELLSWORTH—913
(Central Kansas Society)

DAVIS, GEORGE R. 308 KINGSLEY.67439
472-3121
19 M 1902 44 GS
O'DONNELL, HAROLD F. 412 BLAKE.67439
-
99 M 1902 26 OO
SEITZ JR, JOSEPH E. 308 KINGSLEY.67439
472-3121
22 M 1902 46 FP

EMPORIA—316
(Flint Hills Society)

AMENO, DOUGLAS J. 827 COMMERCIAL.66801
343-6565
46 M 1902 79 OBG
BARNETT, JAMES A. 919 W 12TH.66801
342-2521
54 M 1902 79 IM
BOSILJEVAC JR, JOSEPH E. 2522 W 15TH.66801
342-4843
51 M 1902 75 TS
BRADLEY, H RUSSELL, 1601 STATE.66801
343-2900
35 M 1902 61 FP
BROCKHOUSE, JOHN P. 1601 STATE.66801
343-2900
31 M 1902 57 IM
BURGESON, FRANK G. 1601 STATE.66801
342-6989
40 M 3005 65 OPH
BUTCHER, THOMAS P. 1128 LAWRENCE.66801
342-0722
05 M 1601 34 GS

CAMPBELL, EDWARD G. 1601 STATE.66801
343-2900
31 M 1902 61 FP
COLOSMITH, OSVALDO C. 1024 W 12TH.66801
342-7047
26 M 1902 58 FP
DAVIS, DAVIO R. 1025 STATE ST APT #2.66801
-

02 M 2101 28 OO
EDWARDS, DAVIO J. 1601 STATE ST.66801
343-1191
43 M 2803 69 ORS
ELLIS, BOBBY J. 1601 STATE.66801
343-2900
51 M 1902 77 IM
FARRIS, RONNIE S. 1601 STATE.66801
343-2900
50 M 3840 75 GS
FRAZIER, RICHARD L. 1005 W 12TH.66801
342-5137
40 M 1902 72 GS
GANN, E LAMONTE, RR #2.66801
-
07 M 2802 37 OO
GARCIA, GOULO C. 919 WEST 12TH AVE.66801
342-2521
32 M 3607 58 IM
GEITZ, JAMES M. 919 W 12TH.66801
342-2521
46 M 1902 72 IM
GINAVAN, DUANE A. 1024 W 12TH.66801
342-5876
35 M 1902 62 FP
GLENN, JAMES N. 1601 STATE.66801
343-1191
43 M 4804 66 ORS
HARVEY, JOHN E. 2506 W 15TH.66801
343-2900
39 M 1902 65 OBG
HAVENHILL II, MARSHALL A. 812 ANDERSON ST.66801
343-6400
35 M 1902 61 OBG
HOPPER, CHARLES R. 25 W 5TH.66801
342-2341
17 M 1902 47 FP
HOWELL, BARBARA JOYCE, 2510 W 15TH.66801
343-7590
45 F 3401 78 PD
KELLING, COLLYER, 1614 E WILMAN.66801
343-7676
50 M 1902 75 P
KNECHT, STEPHEN M. NEWMAN MEMORIAL HOSP.66801
342-7722
44 M 1902 70 R
KRETSINGER OO, W BROCK, 1601 STATE.66801
343-2393
48 M 2878 77 IM
MIGUELINO, OLIVER M. NEWMAN HOSP.66801
343-6800
35 M 74801 57 PATH
MORGAN, JOHN L. 919 WEST 12TH.66801
342-2521
15 M 4101 40 IM
NEIGHBOR, RALPH M. 827 COMMERCIAL ST.66801
343-6565
46 M 1902 72 OBG
NEUER, FREDERICK S. NEWMAN HOSP.66801
342-7722
46 M 3601 71 R
PASTOR, VICTOR HUGO, 1601 STATE.66801
342-7715
43 M 13202 68 U
PIERSON, MARK E. 1024 W 12TH.66801
343-6864
50 M 1902 80 FP
RYAN, W SCOTT, 2510 W FIFTEENTH.66801
343-7590
47 M 1902 73 PD
SCHELLINGER, RICHARD P. 1128 LAWRENCE.66801
342-0722
22 M 3005 49 GS
SNOWBARGER, MARVIN D. 1601 STATE STREET.66801
343-2900
29 M 1902 55 FP
SPEARS, CHESTER A. 12TH & CHESTNUT.66801
343-6800
50 M 2834 76 PATH
TRIMBLE SR, DAVID P. 517 MERCHANT STREET.66801
342-2572
04 M 1902 32 OPH
UNDERWOOD, CHARLES C. 25 WEST 5TH.66801
342-2341
07 M 1902 32 IM

VANDER VELDE, STANLEY L. 1527 BERKELEY ROAD.66801

16 M 1902 43 DD
 WRIGHT, KENDALL M. 1024 WEST 12TH.66801
 343-2376
 45 M 1902 71 FP

ERIE—316
 (Southeast Kansas Society)

BRYAN, EMERY C. RT 2 BOX 171.66733

04 M 1902 32 DD
 HANDSHY, STANLEY E. PO BOX 256.66733
 244-3291
 54 M 1902 79 FP

ESKRIDGE—913
 (Flint Hills Society)

WALKER, WILLIAM H. 2ND & CEDAR.66423

13 M 2401 38 IM

EUDORA—913
 (Douglas County Society)

HOLLADAY, KENNETH R. 101 WEST 10TH.66025
 542-2345
 34 M 1902 58 FP

EUREKA—316
 (Butler-Greenwood Society)

CISKEY, WILLIAM J. PO BOX 310.67045
 583-7401
 47 M 1902 72 FP
 SKAER, STANLEY ALLEN. 100 E 16TH.67045
 583-7486
 40 M 3901 65 GS

FORT SCOTT—316
 (Bourbon County Society)

AKERS, GUY I. 710 W BTH.66701
 223-3100
 20 M 1902 53 FP
 ALDIS, HENRY. 710 W BTH.66701
 223-3100
 13 M 1902 41 DBG
 ALDIS, WILLIAM. 710 WEST BTH.66701
 223-3100
 20 M 1902 44 GS
 BASHAM, JAMES J. 702 MEADOW LANE.66701
 -
 14 M 1902 37 DD
 BENAGE, JOHN F. 821 BURKE.66701
 223-2200
 32 M 1902 58 DBG
 BRAUN, EDWARD W. 710 WEST BTH.66701
 223-3100
 42 M 1902 68 U
 BURKE, JAMES J. 710 W BTH.66701
 223-3100
 35 M 2834 61 IM
 CHOW, STANLEY Y. 1410 S EDDY.66701
 223-2200
 18 M 24222 39 R
 DUNSHEE, CARLYLE M. 710 W BTH.66701
 223-3100
 32 M 1902 57 GS
 GETTLER, DEAN T. 710 WEST BTH.66701
 223-3100
 31 M 1902 57 GS
 GORD, JAMES T. 821 BURKE.66701
 223-2200
 21 M 2802 45 PATH
 GRIMALDI, GARY A. 710 W EIGHTH.66701
 223-3100
 49 M 1902 74 DBG
 IRBY, ADDISON C. 416 S JUDSON.66701
 -
 05 M 1606 28 DD

IRBY, PRATT. 710 WEST BTH.66701

223-3100
 13 M 4705 36 U
 KERR, GERALD F. RR 5.66701

44 M 1902 84 PATH
 MCCANN, PATRICK E. 710 WEST BTH.66701
 223-3100
 28 M 1902 59 IM
 MCKENNA, MICHAEL J. 323 S JUDSON SUITE 120.66701
 223-3950
 38 M 1902 64 FP
 NELSON, T. EUGENE. 710 W BTH.66701
 223-3100
 41 M 1902 69 FP
 NICHOLS, ROBERT R. 102 S JUDSON.66701
 223-4100
 50 M 2803 76 FM
 PARRIS, ROGER D. 102 S JUDSON.66701
 223-4100
 51 M 2803 78 FP
 PHELPS, DAVID WAYNE. 102 S JUDSON.66701
 223-4100
 51 M 1902 76 FP
 RADON, SANFORD P. RT 1 BOX 205B.66701
 223-6029
 40 M 1642 66 R
 REEVES, CHARLES S. 710 W BTH.66701
 223-3100
 37 M 1902 63 IM
 SPENCER, JOHN HAROLD. 710 W BTH.66701
 223-3100
 47 M 1902 74 FP
 WEDDLE, DOUGLAS P. 710 WEST BTH.66701
 223-3100
 43 M 1720 69 FP

FREDONIA—316
 (Southeast Kansas Society)

BACANI, DSWALDD. PO BOX 576.66736
 378-3700
 44 M 74810 70 GS
 BAYLES, HUGH G. PO BOX 30.66736
 378-3412
 25 M 1902 52 FP
 BEAL, RAYMOND J. 310 S 15TH.66736
 -
 12 M 1902 38 DD
 RINDT, PHILLIP L. 432 N SEVENTH.66736
 378-2298
 45 M 1902 71 FP
 SUMNER, RALPH N. PO BOX 537.66736
 378-2311
 31 M 1902 57 FP

FT. LEAVENWORTH — 913
 (Leavenworth County Society)

CHAVALA, SUDARSAN, EYE CLINIC.66027
 684-4157
 43 M 49511 68 DPH

GARDEN CITY—316
 (Southwest Kansas Society)

ARRDYO, ZEFERINO. 2124 ANTLER RIDGE DR.67846
 872-2187
 M 74802 GS
 AUSTIN, JOHN D. 601 N 6TH.67846
 276-2346
 14 M 1601 40 FP
 BEGGS, DAVID F. BOX 1077.67846
 275-9671
 39 M 1902 64 IM
 BIGLER, F. CALVIN. 801 N FIFTH.67846
 275-2141
 31 M 801 57 GS
 BRUND, JAMES W. 1133 KANSAS PLAZA.67846
 276-8201
 42 M 4706 66 FP
 CALBECK, JOHN. 2603 BELMONT PLACE.67846
 275-9671
 50 M 1902 75 IM
 COLLINS, SHARON A. 603 N FIFTH.67846
 275-9671
 51 F 2512 78 PD

DONNELLY, WILLIAM P., 1133 KANSAS PLAZA, 67846
276-8201
M

EICHMORN, FRANK D., BOX 719, 67846

276-8132
25 M 1902 56 FP
FENTON, ROBERT M., 603 N FIFTH, 67846

275-9671
20 M 1902 54 FP
FRY, LUTHER L., ST CATHERINE HOSP., 67846
275-7248

41 M 1902 67 OPH
GARDINER, TED M., 603 N FIFTH, 67846

275-9671
48 M 506 74 PO
GILBERT II, JOHN H., BOX 1077, 67846

275-9671
46 M 1902 70 ORS
GREENWOOD, JAMES F., PO BOX 419, 67846
356-1261

33 M 1611 65 FP
HANSEN, FRANK W., 603 FIFTH, 67846
275-9671

42 M 1902 76 PM
HUNSBERGER, TERRY R., D.O., 602 N THIRD, 67846
275-7128

47 M 2878 73 FP
JACKSON, MICHAEL D., 603 N FIFTH, 67846
275-9671

51 M 4814 76 FP
KALBAC, RICHARD W., BOX 1077, 67846
275-3780

45 M 2803 70 OBG
KOKSAL, TOM, PLAZA MED CENTER, 67846
276-8201

M 1902 FP
LAUDERT, SUSAN E., 2508 E FAIR, 67846

51 F 1902 87
MELIN, BRUCE D., 608 N FIFTH, 67846
275-6111

51 M 5605 77 PATH
MEYERS, STEPHEN, BOX 1077, 67846
275-9671

48 M 2834 70 PO
MILLER, ROBERT E., BOX 1077, 67846

275-9671
26 M 4812 55 GS

MITCHELL, ROBERT H., 603 N FIFTH, 67846
275-3750
M ORS

RODRIGUEZ, PAUL L., BOX 1729, 67846
275-6111
39 M 4706 66 R

SHUSS, JOHN L., 801 N FIFTH, 67846
275-2141
49 M 1902 75 GS

SPIKES, MARION E., 603 N 5TH, 67846
275-9444
26 M 1902 62 FP

TEARE, MAX E., 1007 DAVIS, 67846
276-7689
28 M 1902 54 D

TURNER, JOHN W., 210 E SPRUCE, 67846
276-3292
13 M 1902 39 FP

VACHAL, EVA, 608 N FIFTH, 67846
275-6111
F 1902 PATH

WILEY, HORACE M., BOX 1136, 67846
276-6901
12 M 2802 40 GS

ZELLER, MYRON J., BOX 1077, 67846
275-9671
38 M 1902 64 OM

GARDEN PLAIN—316 (Sedgwick County Society)

LIND II, EDWARD J., 728 BIERMANN, 67050
535-2218
53 M 1902 78 FP

REINHARDT-WU, F., TAISSIA L., PO BOX 273, 67050
-
19 F 91302 42 OO

GARDNER—913 (Johnson County Society)

NIKNIA, SEYED M., 427 W MAIN, 66020
884-8711
38 M 51701 67 GS

REECE, A THOMAS, 427 W MAIN, 66030
884-8711
37 M 1902 63 FP

VILLANUEVA, CESAR L., 427 W MAIN, 66030
764-4467
39 M 74802 65 OBG

WHITAKER, MARK A., 136 E MAIN, 66030
884-7822
53 M 1902 77 PO

GARNETT—913 (Anderson County Society)

DOUGHERTY, THOMAS M., 117 W 6TH, 66032
448-5421
28 M 1902 55 FP

HARRIS JR, CLAIB B., 320 S JAK ST, 66032
448-5431
17 M 1902 44 FP

HENDERSON, DAVID V., 117 W SIXTH, 66032
448-5421
48 M 1902 79 FP

LEITCH, DAVID A., GARNETT MEDICAL CENTER, 66032
448-5421
38 M 1902 63 FP

STEVENS, MILDRED J., 202 W 4TH, 66032
448-5454
23 F 1902 47 FP

STEVENS, ROBERT L., 202 WEST 4TH, 66032
448-5454
23 M 1902 47 FP

GIRARD—316 (Crawford County Society)

FRIGGERI, ROBERT W., 111 N SUMMIT, 66743

724-8723
23 M 1902 51 FP
HALL, WESLEY H., PO BOX 158, 66743

724-6154
25 M 1902 57 FP
WILKINS, JAMES T., BOX 158, 66743

724-6154
51 M FP

GLASCO—913 (Cloud County Society)

HARWOOD, CLAUDE J., PO BOX 428, 67445

568-2245
25 M 1902 55 FP

GOODLAND—913 (Northwest Kansas Society)

AUSTIN, KENNETH D., 520 MAIN, 67735

899-5651
33 M 3005 63 FP
LONG, LLOYD D., 520 MAIN, 67735

899-5651
37 M 1720 63 FP
MCCULLOUGH, ROBERT C., 520 MAIN PO BOX 180, 67735

899-5651
25 M 702 58 GP
MITRA, SUDHEER, 1206 MAIN, 67735

899-5976
F GS
OLSON, CLITUS W., 520 MAIN ST, 67735

899-5651
16 M 3005 48 GS

GREAT BEND—316 (Barton County Society)

ALDERSON, THOMAS W., 3520 LAKIN, 67530

792-5341
50 M 1902 75 FP

BEAHM, ANOL W. 3923 BROADWAY, 67530

793-7827

16 M 1902 43 FP

BEAHM, DONALD E. MED ARTS BLDG. 67530

792-3626

45 M 1902 71 OPH

CAVANAUGH, CLAIR J. C K M C. 67530

792-2617

23 M 1803 47 R

DEGNER, JAMES B. 3515 BROADWAY, 67530

792-2617

31 M 1902 57 R

EVANS, WILLIAM R. 1912 LINCOLN, 67530

-

25 M 1902 53 00

FIESER, CARL W. 3515 BROADWAY, 67530

792-2617

45 M 1902 71 R

GATEND, JOSEPH, 1031 JACKSON, 67530

793-8429

25 M 64901 50 OBG

HILL, LARY MICHAEL, 1017 A JACKSON, 67530

793-8141

51 M 1902 77 FP

HOLT, JOHN M. PO BOX 1328, 67530

793-8429

35 M 1902 61 IM

JONES, EDWARD L. 3515 BROADWAY, 67530

792-2511

35 M 1902 61 PATH

KING, WILLIAM T. 3421 FOREST, 67530

793-3501

35 M 1902 61 OBG

KIRBY, MERLIN G. 3520 LAKIN, 67530

793-3091

31 M 1902 56 GS

KRUEGER, HAVEN C. 1023 JACKSON SQUARE, 67530

792-2163

32 M 1902 61 PD

MCALLASTER, WENDALE E. 2111 FOREST, 67530

793-3591

24 M 1902 54 GS

NIEDEREE, W CURTIS, 3520 LAKIN, 67530

793-3091

30 M 3006 56 GS

POLSON, ROBERT C. BOX A 1422 POLK ST, 67530

793-8414

17 M 1902 42 OPH

PRESTON, RICHARD, PO BOX 1328, 67530

793-8426

42 M 1902 69 IM

REPLOGLE, CHARLES B. 2111 FOREST, 67530

793-3591

27 M 1902 53 FP

RUIZ, CARLOS M. PO BOX 1348, 67530

792-3210

25 M 27501 52 P

SAYLER, JEROME, CENTRAL KS MEDICAL CENTER, 67530

792-2511

20 M 4113 50 PATH

SCHUETZ, PERRY N. 1422 POLK BOX A, 67530

793-8414

45 M 1902 71 OPH

SCHUKMAN, JAY S. 3520 LAKIN, 67530

792-5341

50 M 1902 75 FP

SHIVEL, DAVID G. 3523 FOREST, 67530

793-3523

28 M 1902 55 FP

SMITH, PERRY MILTON, 3520 LAKIN, 67530

792-5341

52 M 1902 77 FP

SWAN, MAJOR MARTIN, 3923 BROADWAY, 67530

792-4540

06 M 1902 43 IM

UNREIN, ROBERT J. 1017A JACKSON, 67530

792-2504

29 M 1902 58 FP

WHITE, CHARLES L. 2412 DOVE TERRACE, 67530

-

06 M 1902 36 00

WIGGS, JAMES W. 1027 JACKSON, 67530

792-1336

36 M 1720 63 N

WIKOFF, DONALD L. 3520 LAKIN SUITE 108, 67530

792-7353

49 M 3005 75 U

YOUN, HWAN, 3515 BROADWAY, 67530

792-2617

48 M 58310 73 OR

ZURITA, MARCOS, BOX 1078, 67530

793-3422

46 M 31901 69 ANES

GREENSBURG—316
(Iroquois County Society)

BRADLEY, J RODERICK, BRADLEY-WALDORF CLINIC, 67054

723-2127

23 M 1902 47 FP

WALDORF JR, MELVIN H. BRADLEY-WALDORF CLINIC, 67054

723-2127

23 M 1902 47 FP

HALSTEAD—316
(Harvey County Society)

AILLON, ALEJANDRO J. HERTZLER CLINIC, 67056

835-2241

39 M 26402 63 TS

BAILEY, COLIN, HERTZLER CLINIC, 67056

935-2241

33 M 35205 59 GYN

BEUGELSOIJK, HENRY PETER, 421 SPRUCE, 67056

835-2241

49 M 1902 74 ANES

BOUDREAUX, VELTIN J. PO BOX 53, 67056

835-2241

37 M 4812 64 R

BURNETT, A DEAN, HERTZLER CLINIC, 67056

835-2241

21 M 1902 52 GS

DECKER, DONALD O. HERTZLER CLINIC, 67056

835-2241

31 M 1902 56 CO

EASTES, GARY DEAN, HERTZLER CLINIC, 67056

835-2241

44 M 4812 71 U

GNAU, FREDRIC B. 803 MAIN, 67056

835-2241

42 M 1902 68 OTO

HAIRE, WILLIAM O. 901 MAIN, 67056

935-2241

49 M 1902 74 HEM

HARMS, WILMER A. THE HERTZLER CLINIC, 67056

835-2241

22 M 1902 56 OPH

HOOFFER, WILFORD D. HERTZLER CLINIC, 67056

835-2241

30 M 1902 55 TS

IGLINSKY, W L. 917 W FOURTH, 67056

835-3548

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KIMMEL, KENNETH K. 4TH & CHESTNUT, 67056

835-2241

52 M 1902 77 IM

MALONE, EUGENE M. HERTZLER CLINIC, 67056

835-2241

23 M 1902 56 IM

MARSH, CONNIE M. HERTZLER CLINIC, 67056

835-2241

47 F 1902 75 IM

MONTGOMERY, LLOYD DAN, HERTZLER CLINIC, 67056

935-2241

43 M 3601 69 P

MONTGOMERYSHORT, RUTH G. HERTZLER CLINIC, 67056

-

10 F 1902 37 ENT

MORTON, JOHN E. 320 WALNUT, 67056

-

99 M 35211 26 00

RESCHLY, RON. 4TH & CHESTNUT, 67056

835-2241

M 3806 ORS

RIZZA, ROBERT G. RT #2, 67056

-

30 M 1201 56 PD

SHAH, SHARFUDDIN, HERTZLER CLINIC, 67056

835-2241

31 M 70401 58 IM

STOFFER, ROBERT P. HERTZLER CLINIC, 67056

835-2241

26 M 1902 48 IM

TEJANO, NEONIL A. HERTZLER CLINIC, 67056

935-2241

43 M 74808 67 ORS

WELCH, JACK W. HERTZLER CLINIC, 67056

-

18 M 1902 51 00

WILSON, H RANDOLPH, HERTZLER CLINIC, 67056

835-2241

20 M 4112 45 GYN

HANOVER—913 (Northeast Kansas Society)

WARREN, LINDA O. BOX 38,66945
325-2240
44 F 1902 70 FP
WARREN, ROGER O. BOX 38,66945
337-2214
31 M 1902 57 GS

HARPER—316 (Tri-County Society)

BELAR, RALPH E. 1019 CENTRAL,67058
896-7313
31 M 3005 60 FP
GARNER, BILLIE L. 121 E MAIN,67058
896-3661
25 M 1902 57 FP

HAYS—913 (Central Kansas Society)

ALBERS, ROBERT C. 2707 VINE,67601
628-3261
48 M 1M
ALLEN, JAMES E. 2707 VINE #6,67601
628-3261
46 M 1902 72 1M
APPLEGATE JR, FRANCIS R. 1010 DOWNING,67601
628-8218
30 M 1902 55 OPH
BULA, RALPH E. 3209A WILLOW,67601
-
12 M 1902 37 00
CARLSON, EARL V. DRAWER 430,67601
628-8221
31 M 3005 56 ORS
CECIL III, JOHN, BOX 833,67601
625-6521
43 M 4804 69 R
COOY, DOROTHY, 2704 WOODROW CT,67601
628-4000
29 F 3607 53 P
COOY, JOHN, 2704 WOODROW CT,67601
628-2871
25 M 401 60 P
COX, ROBERT H. 2507 CANTERBURY RD,67601
628-3051
43 M 1902 70 PD
CRAMM, RUSSELL E. 105 W 13TH,67601
625-8269
30 M 1902 56 GS
DOORSCH, JOHN N. 306 W 38TH,67601
628-6151
54 M 1902 79 FP
DYCK, ERIC LEE, FAMILY PRACTICE CLINIC,67601
628-6151
52 M 1902 77 FP
EDDY, VICTOR M. 105 W 13TH,67601
625-2551
29 M 1902 55 GS
HAIGLER, JAMES P. 234 WEST 11TH,67601
625-2537
13 M 3006 39 GP
HALLING, L WILLIAM, 1300 EAST 13TH,67601
625-5646
27 M 5002 57 PATH
HUTCHISON, GLEN C. 3200 COUNTRY LANE,67601
628-8251
21 M 1902 50 ANES
HUTCHISON, MARC K. 213 CASTILLIAN GARDENS,67601
628-6760
M ANES
KANE JR, WILLIAM M. CANTERBURY CLINIC PA,67601
625-3245
27 M 1001 54 OBG
KELLY, A CHRISTINE, 2707 VINE SUITE 7,67601
628-3217
49 F 2846 77 GS
KIFER, C JAMES, BOX 833,67601
625-6521
45 M 1902 71 OR
LASLEY, MICHAEL B. 2707 VINE SUITE 7,67601
628-3217
45 M 1902 71 GS

LOEB, ELBIE L. 2209 CANTERBURY RD,67601
625-4224
51 M 1902 78 1M
MATTICK, IRVIN H. 2818 N VINE,67601
628-8221
18 M 2802 43 ORS
MC DONALD, KEVIN R. 2209 CANTERBURY,67601
628-6014
52 M 3006 78 U
MYRICK, MICKEY, 2501 CANTERBURY RD,67601
623-6151
42 M 3005 74 FP
NEIL, ROY NEWTON, 1108 MAIN ST S 2018,67601
628-3215
38 M 3005 65 PATH
NEWCOMB, WARD M, 1300 E 13TH, 67601
625-5646
47 M 3005 71 PATH
RAJEWSKI, RICHARD L. 2509 CANTERBURY,67601
628-6151
51 M 1902 76 FP
REYNOLDS, JEFFREY C. 2517 CANTERBURY RD,67601
625-7311
39 M 1902 64 ENT
REYNOLDS, LLOYD W. 504 W 36TH,67601
-
10 M 3840 34 00
RICHARDS, DALLAS LEE, 2209 CANTERBURY RD STE A,67601
625-4224
49 M 1902 76 1M
RUTYGAMLUG, LUECHA, 3004 BROADWAY,67601
628-6175
40 M 89101 68 GS
SILER, EUGENE T. 1010 DOWNING,67601
628-8218
24 M 1902 52 OPH
STADALMAN, ROSS EUGENE, 2707 VINE SUITE 7,67601
628-3217
47 M 1902 73 GS
STUMP, HARL G. 105 W 13TH,67601
-
39 M 1902 65 GS
WATTS, HARRY E. 1010 DOWNING AVE,67601
628-8218
27 M 702 54 OPH
WEBER, WALLACE N. 2707 VINE SUITE 10,67601
628-3231
43 M 1902 69 D
WERTH, DARRELL DEAN, 2209 CANTERBURY RD STE D,67601
628-6014
50 M 1902 75 U
WIEGMAN, HUGH ALAN, BOX 833,67601
625-6521
34 M 1803 60 R
WILCOX JR, HOWARD L. PO DRAWER 430,67601
628-8221
44 M 1902 70 ORS

HAYSVILLE—316 (Sedgwick County Society)

MORGAN, NOVA L. 301 W GRAND,67060
225-1371
20 M 3901 50 FP
SAYEED, BASEER A. 138 STEWART,67060
529-0450
49 M 49504 74 1M

HERINGTON—913 (Dickinson County Society)

BUSTOS, JONAS G. 1005 NORTH B,67449
258-3705
41 M 74810 68 GS
DOZIER, FRED S. 1005 NORTH B STREET,67449
258-2215
10 M 4804 34 FP

HESSTON—316 (Harvey County Society)

DIENER, CLAYTON H. PO BOX 386,67062
327-4122
18 M 1902 54 GS
FRIESEN, FLORENCE V. SHOWALTER VILLA,67062
-
87 F 1611 14 00

HENSON, STEVEN R. 608 RANDOM RD. 67062
 327-4174
 61 M 1902 87
 YODER, VERNON E. ROUTE #1. 67062
 283-2400
 31 M 4802 61 P

HIAWATHA—913
 (Northeast Kansas Society)

DUCKETT, THOMAS G. 201 MIAMI. 66434
 10 M 1902 34 DO
 LARSON, DELBERT L. 314 OREGON. 66434
 742-2161
 30 M 1803 64 FP
 MEIDINGER, RAY. 111 S FOURTH. 66434
 742-2135
 03 M 3005 32 FP
 SINNING, GARY. 314 OREGON. 66434
 742-2161
 49 M 1902 74 FP

HIGHLAND—913
 (Northeast Kansas Society)

HALL JR, ERNEST B. 80X 96. 66035
 442-3341
 53 M 1902 78 FP

HILL CITY—913
 (Central Kansas Society)

REDDY, P JAGANNAOHA. 80 WALNUT DRIVE. 67642
 674-2255
 42 M 49511 66 GS

HILLSBORO—316
 (Marion County Society)

ENS, GERHARD GEORGE. 613 SOUTH MAIN. 67063
 947-5931
 20 M 1902 55 FP
 ENS, PETER. 209 SOUTH MAIN. 67063
 947-3671
 14 M 1902 51 FP
 FRANZ, ROBERT G. 704 S MAIN. 67063
 947-3197
 33 M 3901 59 FP
 LOEWEN, PETER S. SKILLED NURSING UNIT. 67063
 91 M 1902 30 DO

HOISINGTON—316
 (Barton County Society)

FERNANDEZ, HECTOR D. 351 W TENTH. 67544
 653-4944
 41 M 74809 66 GS
 FLANVER, FRANK R. 353 W TENTH. 67544
 653-4138
 43 M 1902 79 FP
 MOORE, ROBERT. 814 NORTH ELM. 67544
 653-2151
 22 M 3901 53 FP

HOLTON—913
 (Shawnee County Society)

MOSEY, ERNEST C. 438 HILLCREST DR. 66436
 09 M 1902 34 DO
 MOSEY, RAY H. 801 IOWA. 66436
 04 M 1902 32 DO

HORTON—913
 (Northeast Kansas Society)

FRANCISCO, EGGARD. PD BOX 6. 66439
 486-2646
 39 M 74802 57 GP
 WALTON, PHILIP D. 1903 EUCLID. 66439
 486-2828
 32 M 1902 63 FP

HOXIE—913
 (Northwest Kansas Society)

NEUENSCHWANDER, JOHN. 1401 QUEEN. 67740
 675-3292
 26 M 2802 51 FP
 NEUENSCHWANDER, JOHN RAND. 700 MAIN. 67740
 675-3292
 47 M 1902 72 FP

HUGOTON—316
 (Seward County Society)

FREDERICK, M F. 1006 S JACKSON. 67951
 544-2784
 20 M 1902 44 FP
 IHRIG, ROGER W. 1006 S JACKSON. 67951
 544-2784
 49 M 1902 74 FP
 LENEVE, ROBERT T. 1006 S JACKSON. 67951
 21 M 3901 46 DO

HUMBOLDT—316
 (Southeast Kansas Society)

LONG, EDWARD E. 8TH & NEW YORK. 66748
 473-2411
 21 M 1902 50 FP

HUTCHINSON—316
 (Reno County Society)

ADAMS JR, MARCUS W. 2101 N WALDRON. 67501
 663-6121
 33 M 3901 59 PD
 ALBRIGHT, JEROLD O. 2101 N WALDRON. 67501
 663-6121
 39 M 1902 66 FP
 BARKER, STANTON L. 2101 N WALDRON. 67501
 663-6121
 54 M 1902 79 FP
 BAUER, THOMAS A. 2101 N WALDRON. 67501
 663-6121
 41 M 1902 67 IM
 BLANK, JOHN N. ROUTE 5 BOX 220. 67501
 07 M 1902 38 DO
 BORRA, MARIO J. 1 WEST 8TH. 67501
 662-1751
 24 M 2401 47 U
 BOS, NORMAN C. 2101 N WALDRON. 67501
 663-6121
 24 M 1611 47 ORS
 BOUDA, DAVIO W. 2101 WALDRON. 67501
 663-6121
 45 M 1902 72 IM
 BRADA, DONALD ROBERT. 308 W 20TH. 67501
 662-6041
 39 M 1902 65 P
 BURGER, J OALE. 2101 N WALDRON. 67501
 663-6121
 21 M 1902 46 FP
 CASEY, JAMES. 2101 N WALDRON. 67501
 663-6121
 42 M 3005 69 PO
 CHERVEN, PHILIP L. 1100 N MAIN. 67501
 662-3364
 45 M 2501 71 PO
 DEPENBUSCH, FRANCIS L. 1708 E 23RD. 67501
 663-7187
 38 M 1902 65 OPH
 DUHON, JOHN MARQUE. 1701 E 23RD. 67501
 665-2126
 47 M 4802 75 DR

ECKART, OE MERLE E. 201 N MAIN #31SA.67501
 662-4921
 14 M 1902 40 P
 FALTER, RICHARD T. 1708 E 23RD ST.67501
 663-7187
 38 M 1902 67 OPH
 FOSS, DANIEL C. 2101 N WALORON.67501
 663-6121
 43 M 1902 69 GE
 GARD, RICHARD A. 1100 N MAIN.67501
 225-7710
 51 M 1720 78 OBG
 GOODPASTURE, WILLARD C. RR 3 BOX 4S.67501
 -
 10 M 1602 36 OO
 GRAVES, KATHRYN. 2101 N WALORON.67501
 663-6121
 49 F 1902 74 O
 HEORICK, KENNETH E. 2101 N WALORON.67501
 663-6121
 27 M 1902 53 GS
 HOLDERMAN, WALLACE O. 2101 N WALORON.67501
 663-4406
 28 M 1902 54 ORS
 JARROTT, JOHN B. 1100 N MAIN.67501
 663-2151
 16 M 1902 40 ORS
 JOHNSON, RANDLE C. 1100 N MAIN.67501
 663-2151
 M 1M
 JOHNSON, RICHARD L. 2101 N WALORON.67501
 663-6121
 M R
 KEPKA, DENNIS J. 3117 S MEADOWLAKE DR.67501
 -
 43 M 56101 77 FP
 KLOSTERHOFF, BRUCE E. 720 N MAIN.67501
 662-6041
 45 M 1611 71 P
 LESSER, OANE A. 2101 N WALORON.67501
 663-6121
 49 M 3901 75 U
 LETTNER, HANS T. BOX 159.67501
 662-7801
 23 M 40716 49 PATH
 LUKENS, OAVIO. 2101 N WALORON.67501
 663-1136
 24 M 2307 48 1M
 MCCOY, CHARLES T. 310 WOLCOTT BLDG.67501
 662-0121
 16 M 1902 41 OPH
 McMULLEN, JOSEPH E. 2101 N WALORON.67501
 663-6121
 33 M 1902 62 GS
 MULL, JOHN C. 2101 N WALORON.67501
 663-6121
 34 M 1902 61 OBG
 NEEL, WILBUR B. 2101 N WALORON.67501
 663-6121
 24 M 1902 59 1M
 NEUSCHAFER, DARREL R. 2101 N WALORON.67501
 663-6121
 48 M 1902 74 OBG
 NUNEMAKER, MARION E. PO BOX 1129.67501
 662-5391
 21 M 1902 46 ANES
 OMOHL, NICHOLAS S. 208 BUCKSKIN RD.67502
 663-6121
 47 M 5605 74 1M
 OPENSHAW, CALVIN R. 2020 N WALORON.67501
 662-0569
 21 M 4901 44 GS
 PAIVE, GEORGE E. 220 W 23RD.67501
 -
 94 M 1606 19 OO
 PEASE, GARY L. 1712 E 23RD.67501
 662-4458
 41 M 3005 67 OTO
 PERKINS, JACK L. 2101 N WALDRON.67501
 663-6121
 24 M 1902 53 FP
 RATE, PEGGY S. 2221 E 56TH.67501
 663-6121
 46 F 1902 73 PO
 RATE, ROBERT R. 2221 E 56TH.67501
 663-6121
 47 M 1902 74 1M
 SCALES, WILLIAM M. PLAZA TOWERS.67501
 -
 00 M 1803 31 OO
 SCHROLL, JACK C. 2101 N WALORON.67501
 663-6121
 24 M 1902 49 OBG

SHEARS, ROBERT M. 1100 N MAIN.67501
 662-3364
 20 M 1902 44 PO
 SMITH, THOMAS WILLIAM. 1712 E 23RD.67501
 662-4458
 43 M 1643 68 OTO
 SPITZER, JEROME S. 1100 N MAIN.67501
 663-2151
 33 M 3005 59 FP
 STAFFORD, ROBERT W. 2101 N WALORON.67501
 663-6121
 43 M 2101 69 1M
 STENSAAS, CARL O. 401 WOLCOTT BLDG.67501
 663-9147
 10 M 1902 38 O
 STOUT, JAMES M. 2101 N WALORON.67501
 663-6121
 29 M 1902 55 FP
 TAYLOR, E J. 1100 N MAIN.67502
 663-2151
 34 M 1902 61 FP
 TISOALE, TERRANCE C. HUTCHINSON CLINIC PA.67501
 663-6121
 36 M 6701 61 ORS
 VON RUEN, WILLIAM J. 500 W 20TH.67501
 662-3306
 26 M 1611 52 GS
 WEIOENSAUL, O V. 2101 N WALORON.67501
 663-6121
 50 M 1902 75 1M
 WILLIAMSON, JOHN. 1100 N MAIN.67501
 669-0191
 35 M 2501 60 ORS
 WORTMAN, JACK A. 2101 N WALORON.67501
 663-6121
 34 M 1902 62 1M

INDEPENDENCE—316 (Southeast Kansas Society)

ATWOOD, LARRY C. 800 W MYRTLE.67301
 331-8610
 54 M FP
 BAIR, ALBERT E. PO BOX 925.67301
 -
 16 M 1902 44 OO
 BARBERA, PORTER E. 800 WEST CHESTNUT.67301
 331-4400
 19 M 4707 46 FP
 CHAPPUIE, WILLIAM G. 800 CHESTNUT WEST.67301
 331-5440
 24 M 1902 51 FP
 EMPSON, CHARLES L. 400 N 14TH.67301
 331-6019
 37 M 1902 68 FP
 KNUTH, KENNETH L. 2900 TERRA VISTA.67301
 331-2200
 22 M 1902 50 R
 MYERS JR, EARL B. BOX 548.67301
 331-3420
 32 M 2803 64 GS
 PATEL, V C. PO BOX 883.67301
 331-2725
 50 M 49576 74 ORS
 PHIPPS, RONNY G. PO BOX 843.67301
 331-7901
 54 M 512 80 FP
 PRENTICE, WALTER B. PO BOX 628.67301
 331-8650
 29 M 1643 56 OR
 ROBINSON, EDGAR L. 601 N FOURTH.67301
 -
 15 M 1902 42 OO
 STACEY, KIMBALL. 209 N SIXTH.67301
 331-6350
 M 1M
 SUTTON, ROBERT E. PO BOX 1003.67301
 331-7700
 46 M 1902 72 FP
 TONG, ROLAND M. PO BOX 425.67301
 331-6573
 49 M 74801 72 GS

IOLA—316 (Allen County Society)

COPENING, TELL B. 305 N WASHINGTON.66749
 365-2134
 43 M 1902 69 FP

OETAR, GEORGE F. 219 W MADISON.66749
 365-5174
 28 M 1902 57 GS
 OICK, WILLIS G. BOX 826.66749
 365-2131
 13 M 512 41 GS
 HANSON, DAVID C. 501 E MADISON BOX 714.66749
 365-2131
 46 M 512 73 FP
 LEVSKI JR, FRANCIS X. 206 S JEFFERSON.66749
 365-3901
 26 M 1606 49 FP
 MYERS, W EUGENE. 211 S STREET.66749
 365-3732
 12 M 1902 46 FP
 PEES, GERA_O B. 219 W MADISON.66749
 365-5175
 15 M 1902 43 GS
 SCHMAUS, _Y_E F. 1020 N JEFFERSON.66749
 -
 99 M 1803 26 00
 WOLFE, BRIAN O. 305 N WASHINGTON.66749
 365-2134
 M FP

JETMORE—316
(Ford County Society)

O'SHEA, JAMES G. BOX 545.67854
 357-8321
 18 M 3901 48 FP

JEWELL—913
(Mitchell County Society)

PLOWMAN, CARL W. .66949
 -
 99 M 1606 26 FP

JUNCTION CITY—913
(Geary County Society)

BOLLMAN, CHARLES S. PO BOX 397.66441
 762-4575
 41 M 3901 66 GS
 BRETHOUR, LESLIE J. 1106 ST MARYS RD.66441
 238-4151
 13 M 3006 39 FP
 BUNKER JR, HERBERT L. 1106 ST MARYS RD.66441
 238-5131
 20 M 1606 45 FP
 COPELAND, GARY A. GEARY COMMUNITY HOSP.66441
 762-2387
 42 M 1902 68 R
 CRAIG, THOMAS A. 1106 ST MARYS RD STE 102.66441
 762-4255
 53 M 1902 78 1M
 LABHSETWAR, S A. MEDICAL ARTS BLDG.66441
 762-4147
 39 F 49528 64 OBG
 MACE, RONALD O. PO BOX 163.66441
 762-4884
 42 M 3901 74 FP
 MINNICK, CHARLES V. 435 W CHESTNUT.66441
 -
 08 M 2105 35 FP
 O'DONNELL, HARRY E. 1106 ST MARYS RD.66441
 762-4947
 14 M 4113 42 FP
 PATEL, MAHENDRA N. 1106 ST MARYS RD STE 206.66441
 762-2327
 48 M 35207 74 1M
 SCOTT, ALEX. 507 WEST 6TH.66441
 238-2518
 23 M 5605 48 FP
 WRIGHT, STANLEY E. 1106 ST MARYS RD.66441
 762-4884
 47 M 3901 74 FP

KANSAS CITY—913
(Wyandotte County Society)

ABDOU, NABIH I. KU MEDICAL CENTER.66103
 588-6586
 34 M 33002 58 A

ADKISSON, WAYNE O. 3824 BOOTH APT 3.66103
 -
 61 M 1902 87
 AMHEMANN, JANET L. 4464 FISHER.66103
 -
 57 F 1902 86
 ALDERMAN, LILLIAN C. 1928 W 36TH.66103
 -
 53 F 1902 84
 ALEXANDER, CHARLES E. TWO GATEWAY CENTER #917.66101
 321-6670
 43 M 401 70 OBG
 ALEXANDER, CLYDE W. 1514 NORTH 5TH.66101
 281-4380
 96 M 4707 23 FP
 ALGIE, WILLIAM H. 907 N 7TH.66101
 281-2929
 02 M 1902 27 1M
 ALIFANO, CONNIE L. 3730 CAMBRIDGE.66103
 -
 59 F 1902
 ALLEGRE, ANA. 1225 N 78TH.66112
 788-7099
 50 F 1902 77 1M
 ALLEN, KEITH B. 3126 EATON.66103
 -
 60 M 1902 86
 ALLEN, TIMOTHY E. 9201 PARALLEL PKWY.66112
 651-7195
 49 M 1902 76 R
 ALLEN JR, WILLIAM R. 9201 PARALLEL.66112
 334-4110
 46 M 1902 78 R
 AMARE, MAMMD, KU MEDICAL CENTER.66103
 588-6077
 36 M 60501 61 1M
 APPELBAUM, JAMES S. 4140 800TH PL APT 7.66103
 -
 63 M 1902 86
 ARAKAWA, KASUMI, KU MED CENTER.66103
 588-6670
 26 M 57211 53 ANES
 ARENAL, ANGELA C. BETHANY HOSPITAL.66102
 281-8839
 38 F 35204 60 ANES
 ASHER, MARC A. K U MED CENTER.66103
 588-6130
 36 M 1902 62 DRS
 ATKINS JR, FLOYD L. K U MED CENTER.66103
 588-6015
 43 M 5104 69 CO
 BARADA, JAMES M. 3928 BOOTH.66103
 -
 89 M 1902 85
 BARNES, RICHARD E. 1854 S 32ND.66106
 -
 58 M 1902 84
 BARR, RDBIN R. 1919 FEDERAL.66103
 -
 55 F 1902 84
 BASS JR, LEWIS N. 1975 N 5TH.66101
 321-2320
 21 M 1902 45 PD
 BATNITZKY, SOLOMON, K U MED CENTER.66103
 588-6835
 40 M 83601 64 DR
 BEACH, ROBERT L. 1901 W 42ND.66103
 -
 45 M 1902 86
 BEARCE, SHARON C. 3932 ADAMS APT 18.66103
 -
 58 F 1902 87
 BEARDEN, DENNIS E. 2424 W 40TH APT #29.66103
 -
 59 M 1902 87
 BECKER, LESLIE E. 600 NEBRASKA.66101
 342-4010
 23 M 1003 46 U
 BEJAR, JOSE M. 3838 RAINBOW BLVD APT 609.66103
 588-6970
 46 M 31901 74
 BELDEN, MARY J. 3921 BOOTH #11.66103
 -
 61 F 1902 87
 BELL, GREGORY A. 3702 CAMBRIDGE.66103
 -
 59 M 1902 86
 BELZ, MICHAEL K. 4015 BOOTH.66103
 -
 61 M 1902 87
 BENSON, KIRK T. KU MED CENTER.66103
 588-6670
 54 M 1902 79 ANES

BERGIN, JAMES J. BETHANY MED CENTER, 66102
281-8767
28 M 2407 S4 IM
BERRIDGE, DEBRA L. 2424 W 40TH APT 21, 66103

60 F 1902 87
BETTIN, BRYCE D. 3821 SPRINGFIELD #10, 66103

60 M 1902 87
BETTY, JANE M. 4312 ADAMS, 66103

S1 F 1902 87
BICHLMEIER, FRANKLIN G. 1SS S 18TH, 66102
371-6800

33 M 1902 S8 COS
BIXLER II, THOMAS J. KU MED CTR CARDIOVAS SURG, 66103
588-6107

47 M 1205 74 COS
BLACK, DAVID L. 4448 FISHER, 66103

S8 M 1902 84
BLOMQUIST, GLENDA L H. 201 N 82ND, 66112

S6 F 1902 85
BODENSTEINER, DAVID C. K U MED CENTER, 66103
588-6077

51 M 1903 76 IM
BOGGAN, MICHAEL D. 155 S 18TH, 66102
371-6800

40 M 1201 67 COS
BOLING, JAMES M. 4127 THOMPSON APT 5, 66103

S8 M 1902 84
BOLINGER, ROBERT E. KU MEDICAL CENTER, 66103
588-6022

19 M 1902 43 END
BOSILEVAC, FRED N. 155 S 18TH, 66102

16 M 1902 44 OPH
BRACKETT JR, CHARLES E. KU MED CENTER, 66103
588-6117

20 M 3501 44 NS
BRANIECKI, MARYLEE A. 4130 EATON, 66103

59 F 1902 86
BRAUN, STEVEN D. 4101 FRANCIS, 66103

61 M 1902 87
BRILLHART, MAXINE T. 1610 WASHINGTON BDU. EVARD, 66102
321-4800

15 F 1902 50 FP
BROOKS, WILLIAM HENRY. 155 S 18TH, 66102
371-4343

49 M 1902 74 R
BROSE, WILLIAM G. 3741 SPRINGFIELD, 66103

58 M 1902 84
BROWN, JOHN V. 3728 CAMBRIDGE, 66103
432-5610

59 M 1902
BROWN, MELINDA K. 3804 800TH #7, 66103

61 F 1902 87
BROWN, MICHELLE R. 2100 W 43RD, 66103

56 F 1902 86
BROWN, ROBERT L. 4200 LLOYD, 66103

60 M 1902 87
BUBB, STEPHEN K. 155 S 18TH, 66102
371-6802

48 M 1902 74 ORS
BURGER, WILLIAM E. 355 NEW BROTHERHOOD BLDG, 66101
371-1017

21 M 3006 S1 GS
BUTTERFIELD, MARI ANNE. KU MED CENTER, 66103
588-6670

52 F 1902 77 A4E5
CALDERON, JAIME. 4631 ORVILLE SUITE 201, 66102
287-5556

39 M 26401 66 CO
CALKINS, JOHN W. KU MED CENTER, 66103
588-6236

S1 M 1902 76 DBG
CAMERON, WILLIAM J. KU MEDICAL CENTER, 66103
588-6200

29 M 2501 S4 JBG
CARLIN, JAMES WILLARD. KU MED CENTER, 66103
588-6670

S1 M 1902 76 ANES
CARPENTER, PAUL R. 155 S 18TH SUITE 105, 66102
371-6800

24 M 1902 50 GS

CARRD, TONY L. 4338 LLOYD, 66103

S7 M 1902
CARTY, RUSSEL W. 3900 800TH #9, 66103

S3 M 1902
CASTEEN, JOHN A. 3909 800TH #3, 66103

60 M 1902 87
CATO, TERI A. STUDENT UNION BLDG RM 272, 66103

60 F 1902 87
CAVANAUGH, TIMOTHY B. 3909 ADAMS, 66103

60 M 1902 86
CHALIAN, ALEXANDER R. 2648 MINNESOTA AVENUE, 66102

03 M 3509 37 OO
CHANG, C H JOSEPH. KU MED CENTER, 66103
588-6807

29 M S8301 S3 R
CHENG, MEI Y. KU MED CENTER STUDENT, 66103

46 F 1902 86
CHIN, CRAIGHTON. 3030 EATON, 66103

S4 M 1902 87
CHIN, TOM D. KUMC - HUMAN ECOLOGY DEPT, 66103
588-7175

22 M 2501 43 IO
CHD, CHENG T. K U MED CENTER, 66103
588-6336

37 M 38501 62 PO
CHONKO, ARNOLD M. KU MED CENTER, 66103
588-6076

43 M 3840 69 NEP
CLARK, CHUCK. 3909 ADAMS, 66103

60 M 1902 86
CLARK, FRANCIE H. 3921 800TH #7, 66103

S7 F 1902 87
CLAWSON, D KAY. K U MED CENTER, 66103
588-1207

27 M 2401 S2 ORS
COADY, MARY ANN. 3915 800TH APT 7, 66103

S9 F 1902 86
COALE, LLOYD H. 5020 GREELEY, 66104

13 M 1902 43 OO
COOK, JAMES D. KU MED CENTER, 66103
588-6077

35 M 6505 60 HEM
COOK, THEODORE R. 3901 800TH APT 1, 66103

61 M 1902 87
COOKE, ALLAN R. KUMC DEPT OF MED, 66103
588-6990

36 M 14303 S8 GE
COONFIELD, JAMES W. 1428 S 32ND, 66106
384-1630

45 M 1902 78 FP
COOPER, CATHY N. KUMC BOX 35, 66103

62 F 1902 86
COWLEY JR, BENJAMIN D. 4125 THOMPSON RD 12, 66103

S6 M 4804 81 IM
COX III, IRA L. 155 SOUTH 18TH, 66102
371-4343

43 M 1902 68 OR
CROCKETT, CHARLES A. 155 S 18TH, 66102
342-2200

19 M 401 44 OPH
CULP, LOUIS M. 1645 WASHINGTON BLVD, 66102
371-1077

24 M 1902 S3 FP
CZAPANSKY, DESIREE K. 3838 RAINBOW APT 208, 66103

S9 F 1902
DADKHAH, NAHER. 4124 THOMPSON #5, 66103

S7 M 1902 87
DARR, RICHARD B. 320 TERRACE TR WEST, 66106
676-2097

42 M 3401 70 IM
DAVIS, BRADLEY E. 3808 800TH APT 8, 66103

60 M 1902 86
DAVIS, CHRISTOPHER G. 219 MURDOX BLDG, 66101
321-9313

09 M 1902 39 FP

DAY, HUGHES W. BETHANY MED CENTER.66102

281-8856

15 M 1902 39 IM

DE SNEY, ARTHUR AUGUST, KU MED CENTER - DIAG RAD.66103

588-6800

47 M 2501 72 DR

DE VRIES, PIETER A. KU MED CTR PEDIATRIC SURG.66103

588-6180

21 M 511 47 PDS

DEITZ, MICHAEL R. 155 S 18TH.66102

342-2222

32 M 4101 58 DPH

DEMOTT, WAYNE R. PROVIDENCE-ST MGT HLTH CT.66112

334-2500

34 M 4002 59 PATH

DEWBERRY JR. GLENN P. KU MED CTR DEPT DF FP.66103

588-1908

50 M 3901 76 FP

DIALLO, GASTON I. 600 NEBRASKA.66117

281-2888

35 M 86905 64 GE

DICK, ARTHUR R. KU MEDICAL CENTER.66103

588-6985

34 M 2301 65 N

DIEDERICH, DENNIS A. K U MEDICAL CENTER.66103

588-6981

36 M 2834 61 IM

DDUBEK, DEBBIE L. 3828 8DDTH APT 4.66103

-

58 F 1902 86

DUGAN, DAVID L. 1918 W 37TH.66103

-

56 M 1902 87

DUJDVNE, CARLOS A. K U MED CENTER.66103

588-6026

37 M 13201 61 CP

DULIN, JOSE I. 6013 LEAVENWORTH RD.66104

299-0089

51 M 84711 75 IM

DUNN, MARVIN I. KU MED CENTER.66103

588-6015

27 M 1902 54 CD

EDWARDS, SHELLEY J. 4120 THOMPSON #9.66103

-

58 F 1902 87

EGEA, FERNAND M. 8919 PARALLEL PARKWAY.66112

334-3400

37 M 13206 62 N

EISEMANN, ALLAN D. 3932 ADAMS #21.66103

-

59 M 1902 86

ELLIS, LAVELLE A. 7208 PITKIN.66111

-

60 F 1902 86

FABIAN, CAROL J. K U MED CENTER.66103

588-6029

46 F 1902 DN

FAIDLEY, CHERYL K. 3730 CAMBRIDGE.66103

-

58 F 1902 85

FITZPATRICK, M ROBERT. 1610 WASHINGTON BLVD.66102

321-4800

20 M 1803 44 FP

FLDERSCH, HUBERT M. 8919 PARALLEL PARKWAY.66112

371-2020

35 M 1902 35 JBG

FLUTER, GEDRGE G. 3732 CAMBRIDGE.66103

-

57 M 1902 87

FORET, JOHN D. KU MED CENTER.66103

588-6147

26 M 1602 53 U

FOX, DEANNA K. K U MED CENTER.66103

588-6670

48 F 1902 74 ANES

FOX, HOWARD A. K U MED CENTER.66103

588-6337

33 M 3501 62 PD

FOX, REBECCA R. 3838 RAINBOW APT 601.66103

-

61 F 1902 87

FRANCISCO, W DAVID. 155 S 18TH.66102

371-6802

21 M 1902 44 DRS

FREDRICKSON, ODREN D. 8DK 56 STUDENT KUMC.66103

-

55 M 1902 86

FRIESEN, STANLEY R. KU MED CENTER.66103

588-6108

18 M 1902 43 GS

GABRIELLI JR, WILLIAM F. 4117 ADAMS #101.66103

-

55 M 1902 87

GALLER, GREG WAYNE. 3921 8DDTH #6.66103

-

58 M 1902 86

GERJARUSAK, PRAPAS. 8919 PARALLEL SUITE 208.66112

788-9604

46 M 89101 71 IM

GILHDUSEN, FREDERIC M. 1029 N 32ND.66102

281-5252

40 M 1902 66 DRS

GILLENWATER, DAVID T. 2522 W 42ND APT 3.66103

-

60 M 1902 86

GILLET, MARK L. 3902 8DDTH APT 2.66103

-

33 M 1902 87

GIRDN JR, LOUIS T. K U MED CENTER DEPT DF N.66103

588-6970

43 M 2802 68 N

GLDTZBACH, ROBIN K. 4131 EATON.66103

-

55 F 66103 85

GLOVER II, RICHARD M. 3900 8DDTH APT 10.66103

-

56 M 1902 87

GODFREY, WILLIAM A. KU MED CENTER.66103

588-6600

38 M 1902 65 JPH

GDERTZ, KENNETH K. 222 HC MILLER KUMC.66103

588-6311

50 M 1902 75 PD

GDERTZ, LEO R. 155 S 18TH ST.66102

371-4343

22 M 1902 52 R

GOLDBERG, JOHN M. 3838 RAINBOW.66103

-

60 M 1902 87

GONTERD, ELIZABETH K M. 3130 EATON.66103

-

59 F 1902 87

GODDWIN, DONALD W. KU MED CENTER.66103

588-6402

31 M 1902 64 P

GODDWIN, JOHN A. 1104 SUNTREE PL 1803.66103

-

60 M 1902 86

GORTON, MICHAEL E. 2559 W 46TH.66103

-

61 M 1902 86

GOTO, HIRDSHI, KUMC - ANES DEPT.66103

588-6670

42 M 57241 67 ANES

GRABAU, GUY M. 3121 HAGEMANN.66106

-

54 M 1902 86

GRABER, KARDLYN M. 2520 W 39TH APT 1A.66103

-

61 F 1902 87

GRADY, KENNETH L. 155 S 18TH.66102

321-3844

36 M 1902 69 P

GRANTHAM, JARED J. K U MED CENTER.66103

588-6075

36 M 1902 62 NEP

GREENBERGER, N J. KU MEDICAL CENTER.66103

588-6001

33 M 3806 59 IM

GRELINGER, BART A. 3934 SPRINGFIELD.66103

-

M 1902 87

GRUENDEL, RICHARD A. 1029 N 32ND.66102

281-5252

29 M 1902 55 DRS

GRUENDEL, VIRGINIA T. 6926 GARFIELD.66102

299-2787

30 F 1902 55 FP

GUARDIA, DAVID K. KU MED CENTER DEPT OB/GYN.66103

588-6238

52 M 1902 77 D8G

GUMUCIO, MARID L. 6013 LEAVENWORTH RD.66104

299-2069

30 M 64901 63 IM

HALL III, THOMAS BRYAN, KU MED CENTER.66103

588-6400

43 M 2802 69 P

HAM, ROBERT E. 4125 THOMPSON APT 10.66103

-

53 M 1902 86

HAMILL, JOHN M. 3915 8DDTH APT 3.66103

722-5635

59 M 1902

HANCOCK, ALAN C. 9201 PARALLEL.66112

299-1474

35 M 1902 65 FP

HANSON, FREDERICK, K U MED CENTER, 66103

588-6044

48 M 506 74 IM

HARA, GLENN S, KU MED CENTER, 66103

588-6200

43 M 514 69 D8G

HARDIN, CREIGHTON A, KU MED CENTER, 66103

588-6106

18 M 5605 43 GS

HARDTEN, DAVID R, 3909 800TH APT 12, 66103

-

61 M 1902 87

HARPER, DIANE M, 4136 800TH, 66103

-

58 F 1902

HARRIS, JO ANN SPEIGEL, K U MED CENTER, 66103

588-2773

50 F 3901 76 PD

HART, KELLY Z, 155 S 18TH, 66102

371-4343

50 M 1902 75 DR

HARTMAN, CHARLES R, K U MED CENTER, 66103

588-6111

37 M 1902 66 IM

HARTMAN, GERALD V, KUMC, 66103

588-6815

20 M 1902 45 TR

HELLMAN, DAVID W, 1100 COUNTY LINE APT 24, 66103

-

59 M 1902 87

HENDRICKS, WILLIAM J, 1901 W 42ND, 66103

-

60 M 1902 86

HENKE, JEFFREY L, 4117 ADAMS APT 212, 66103

-

59 M 1902 86

HENNING JR, HAROLD JOHN, 4116 800TH, 66103

-

55 M 1902 82 D8G

HERMRECK, ARLD S, K U MED CENTER, 66103

588-7232

38 M 1902 65 GS

HETTINGER, MICHAEL E, KU MED CENTER, 66103

588-6600

46 M 4706 75 DPH

HIEBERT, JOHN M, KU MED CTR PLASTIC SURG, 66103

588-6143

42 M 2405 67 PS

HINMAN, MARK W, 3838 RAINBOW, 66103

-

59 M 1902 86

HINTHORN, DANIEL R, KU MED CENTER, 66103

588-6035

41 M 1902 67 IM

HOADLEY, WILLIAM D, KU MED CENTER-MEDICINE, 66103

588-3974

31 M 1902 56 IM

HOBSDON, LISA L, 3804 800TH #7, 66103

-

61 F 1902 87

HODGES, GLENN R, K U MED CENTER, 66103

588-6035

41 M 1602 67 1D

HODGSON, JAMES F, 8919 PARALLE, 66112

299-8000

45 M 4813 77 D8G

HOLDCRAFT, JACQUELYNE, 4631 ORVILLE SUITE 203, 66102

321-1161

36 F 2105 53 ENT

HOLMES, FREDERICK F, KUMC, 66103

588-6005

32 M 5404 57 IM

HOLMES, GRACE E, KUMC, 66103

588-6325

32 F 5404 57 PD

HOLMES, JOHN A, 155 S 18TH, 66102

621-1188

47 M 1902 77 IM

HON, DAVID E, 4122 THOMPSON APT 24, 66103

-

60 M 1902 86

HOBSTETLER, ROBERT W, 4316 LLDYD, 66103

-

55 M 1902 87

HUERTER, QUENTIN C, WYANDOTTE MED BLDG STE226, 66112

299-8800

31 M 1902 59 OPH

HURWITZ, ARYEH, KU MED CENTER, 66103

588-6060

36 M 2802 61 IM

IBARRA, RICHARD C, 754 PACIFIC, 66101

342-3969

26 M 64902 57 FP

INGRAM, JDMH E, 1428 S 32ND, 66106

384-1630

24 M 3006 56 FP

JACOBSS, DANIEL H, 3127 EATON, 66103

-

61 M 1902 86

JACOBS, DAVID S, 8929 PARALLEL PARKWAY, 66112

596-4725

31 M 2501 56 PATH

JACOBSS, RAE R, K U MED CENTER, 66103

588-6163

36 M 3506 62 ORS

JAHANIAN, DARYDUSH, 8919 PARALLEL PARKWAY, 66112

371-2020

40 M 51701 64 D8G

JAYARAM, MARANDAPALLI R, 8919 PARALLEL, 66112

492-6200

42 M 49509 65 PD

JEWELL, WILLIAM R, KU MED CENTER, 66103

588-6112

35 M 1611 61 GS

JOHNSON, BRUCE E, KU MED CENTER, 66103

588-6000

50 M 514 76 IM

JOHNSON, CYNDA A, KU MED CTR FAMILY PRACTIC, 66103

588-1908

51 F 514 77 FM

JOHNSON, JDMH E, BETHANY MED CTR, 66102

281-8815

17 M 4706 43 PATH

JONES JR, HERMAN H, 600 NEBRASKA, 66101

342-4010

25 M 4707 54 GS

JORDAN, JANET C, 3900 ADAMS APT 8, 66103

-

53 F 1902 87

KALIVAS, JAMES T, KU MED CENTER, 66103

588-6028

38 M 502 63 D

KENAGY, ROBERT S, 2424 W 40TH APT 32, 66103

-

57 M 1902 87

KENNEDY, JAMES A, K U MED CENTER, 66103

588-6000

35 M 2834 61 IM

KEPES, JOHN J, K U MED CENTER - PATH DEPT, 66103

588-7076

28 M 47301 52 PATH

KERBY, GERALD R, KU MED CENTER, 66103

588-6044

32 M 1902 58 PUD

KESTENBAUM, THELDA M, K U MED CENTER, 66103

588-6028

48 F 5101 73 D

KEYTER, IVAN C, 4323 CAMBRIDGE, 66103

-

60 M 1902 87

KIM, JONG M, KUMC, 66103

588-6670

40 M 58302 64 ANES

KIM, SUCHA, 4142 800TH PLACE #8, 66103

-

51 F 1902 87

KING, CHARLES R, K U MEDICAL CENTER, 66103

588-6200

47 M 1902 71 D8G

KING, TERESA M, 4309 ADAMS, 66103

384-5237

59 F 1902

KIRCHNER, FERNANDO R, 155 S 18TH SUITE 270, 66102

371-7333

30 M 64901 55 OTJ

KLAUMANN, MICHELLE A, 3808 800TH #7, 66103

-

59 F 1902 87

KOVAC, ANTHONY L, KU MED CENTER ANES DEPT, 66103

588-6670

52 M 1902 77 ANES

KRANTZ, KERMIT E, KU MED CENTER, 66103

588-6201

23 M 1606 48 D8G

KUEBLER, KEVIN M, 155 S 18TH, 66102

371-6800

50 M 2101 75 GS

KUMMER, ANTHONY J, 3821 SPRINGFIELD APT A, 66103

-

51 M 1902 87

KYNER, JOSEPH L, KU MED CENTER, 66103

588-6048

34 M 1902 60 END

LAI, CHI-WAN, K U MED CENTER DEPT OF N, 66103

588-7189

44 M 38502 69 N

LAING, ROBERT R. 155 S 18TH ST.66102
371-4301
37 M 1643 61 SE
LAWHEAD, JEFF D. 3033 PUCKETT RD #18.65103
-
57 M 1902 84
LAWHORN, CHARLTON D. 3909 800TH APT 9.66103
-
54 M 1902 85
LAWHORN, STEPHANIE LU. 3909 800TH APT 9.66103
722-0691
54 F 1902
LAWTON, STEVEN K. 3932 ADAMS APT 20.66103
-
61 M 1902 87
LAWWILL, THEODORE, K U MED CENTER.66103
588-6605
37 M 4705 61 OPH
LAYBOURNE JR, PAUL C. KU MED CENTER.66103
588-6475
19 M 3509 44 CMP
LEAR, REX V. 3218 EATON.66103
-
60 M 1902 86
LEE, JAE M. 155 S 18TH #105.66102
677-3555
40 M 58302 65 GS
LEE, KYO R. KJMC.66103
588-6800
33 M 58302 59 R
LEE JR, JAMES G. 5739 METCALF CT.66202
371-2330
18 M 1902 44 OBG
LEMOINE JR, ALBERT N. K U MED CENTER.66103
588-6600
18 M 2802 43 OPH
LEO, WILLIAM A. K U MED CENTER.66103
588-6109
22 M 1902 48 GS
LEVINE, ERROL. < U MED CENTER.66103
588-6800
41 M 83601 64 DR
LIBEL, ROY. 3740 800TH APT 6.66103
-
53 M 1902 86
LIEBERMAN, BRUCE IRWIN. KU MED CENTER.66103
588-6300
49 M 3819 74 PD
LINDSLEY, CAROL B. K U MED CENTER.66103
588-5907
41 F 5404 68 PD
LINDSLEY, HERBERT B. K U MED CENTER.66103
588-6008
40 M 1902 66 RHU
LINN, CATHERINE P. 155 S 18TH.66102
788-9797
52 F 1902 77 OBG
LINSHAW, MICHAEL A. K U MED CENTER PED DEPT.66103
588-6323
40 M 4109 66 PD
LIU, ALBERT T. 8919 PARALLEL.66112
798-9797
49 M 1902 79 OBG
LIU, CHIEN, K U MED CENTER.66103
588-6035
21 M 24217 47 ID
LLOYD, HARVEY L. PD 8DX 6037.66106
-
08 M 1803 36 FP
LOTITO, CARLOS A. 4631 ORVILLE.66102
596-1185
29 M 13201 56 IM
LOVETT, BRENT R. 3812 800TH APT 6.66103
-
59 M 1902
LOWMAN, JAMES T. KUMC.66103
588-6340
31 M 401 58 PD
LUBETICH JR, JOHN F. 4117 ADAMS APT 312.65103
-
57 M 1902 87
LUKERT, BARBARA P. KU MED CENTER.66103
599-6048
34 F 1902 60 END
LYLE, LINDA S. 4405 CAMBRIDGE.66103
-
53 F 1902 87
LYNCH, SEAN R. KU MED CENTER.66103
588-6031
38 M 83601 61 HEM
MACARTHUR, RICHARD IAN. <UMC 39TH AT RAINBOW.66103
588-6196
46 M 1902 73 GS

MADDEN, CATHERINE E. 3934 SPRINGFIELD.66103
-
83 F 1902 87
MAGRINA, JAVIER F. K U MED CENTER OBG DEPT.66103
588-6244
49 M 84701 68 OBG
MALONE, DAVID G. 3952 ADAMS #24.66103
-
60 M 1902 86
MANGOLD, JOEL VOYCE. KU MED CENTER.66103
588-6670
50 M 1902 76 ANES
MANI, MANI M. KUMC.66103
588-6142
37 M 49527 60 PS
MARGOLIS, MICHAEL T. 3828 ADAMS.66103
-
57 M 1902 87
MARPLES, BRADLEY #. 3904 ADAMS.66103
-
56 M 1902
MARSH, ALICE GARRISON, K U MED CENTER.66103
588-6390
24 F 3545 49 PD
MARSHALL, ROGER W. 3728 800TH #8.66103
-
60 M 1902 87
MARTIN, JOSEPH P. 9201 PARALLEL.66112
334-1515
49 M 1902 74 IM
MARTIN, NORMAN L. K U MED CENTER.66103
588-6800
36 M 1902 62 DR
MARTIN, RONALD L. KU MED CENTER PSYCHIATRY.66103
588-6412
45 M 1606 71 P
MASSOTH, SUE V. 3739 CAMBRIDGE.66103
722-0682
52 F 1902 86
MASTERS, FRANCIS W. KU MED CENTER.66103
588-6142
20 M 3545 45 PS
MATHEWSN, HUGH S. KUMC.66103
588-6675
21 M 1902 44 ANES
MATTIDLI, LEDNE. KUMC.66103
598-6311
32 M 56115 56 PDC
MC ALLISTER, SCOTT H. 3909 ADAMS.66103
-
59 M 1902 86
MC GOWAN, CATHERINE C. 4015 800TH.66103
-
60 F 1902 87
MC PHEE, MARK S. KU MED CENTER-MEDICINE.66103
588-6001
51 M 1902 76 IM
MC CARTHY, ROBERT P. 155 S 18TH.66102
342-7233
25 M 2834 53 U
MCCUNE, MARK A. 8919 PARALLEL PKWY.66112
492-6200
52 M 1902 77 D
MCKITTRICK, RICHARD. 4117 ADAMS #314.66103
-
59 M 1902 86
MEBUST, WINSTON K. KU MED CENTER.66103
588-6146
33 M 5404 58 U
MEDHAT, MOHAMED A. KU MED CENTER REHAB MED.66103
588-6798
32 M 33002 54 RM
MEEK JR, JOSEPH C. KU MED CENTER.66103
588-6023
31 M 1902 57 IM
MERFIELD, CHRISTOPHER D. 3900 800TH #8.66103
-
60 M 1902 87
MESINA, ROLANDO R. 1200 N 38TH.66102
371-3829
37 M 74801 61 GS
MILLER, DENNIS W. 600 NEBRASKA SUITE 102.66101
621-4001
49 M 4707 75 OBG
MILLER, KEVIN B. 4132 FISHER #10.66103
-
60 M 1902 86
MILLER, KEVIN E. 4146 800TH.66103
-
59 M 1902 87
MILLIGAN, DONALD B. KU MED CENTER.66103
588-1937
48 M 2307 74 FP

MINER JR, PHILIP B, K U MED CENTER, 66103
588-6990

46 M 702 71 GE
MISKE, STEPHANIE A, 3836 RAINBOW APT 108, 66103

56 F 300S 82 IM
MITCHELL, DEANNA SUE, 1100 COUNTY LINE R 8-9, 66103

60 F 1902 87
MITCHELL, GALEN W, 3133 W 44TH TERR, 66103

53 M 1902 84
MOELLER, DONALD D, 1SS S 18TH, 66102
371-4301

34 M 1902 60 GE
MOORE, JOHN B, KU MED CENTER, 66103

588-6139
51 M 164S 76 PS
MOORE, WAYNE V, K U MED CENTER, 66103

588-6336
42 M 2604 70 PD
MUELLER, MICHAEL A, 3602 RAINBOW #306, 66103

60 M 1902 86
MULLEN SR, CLIFFORD J, 1828 WASH BLVD, 66102

98 M 3006 23 OO
MURPHY, MAUREEN E, 4329 PEARL, 66103

51 M 1902 85
MURPHY, WILLIAM R, 2810 W 42ND, 66103

59 M 1902 87
NAOER, OADKAH, 4124 THOMPSON APT S, 66103

57 M 1902 87
NAZARIO, LILIANA E, 4470 800TH, 66103

57 F 1902 85
NEFF, JAMES R, KU MED CENTER, 66103

588-6198
40 M 1902 66 ORS
NEIGHBOR, ERNEST G, 1420 S 42ND, 66106

06 M 1902 33 FP
NEIGHBOR, ERNEST H, 1420 S 42ND, 66106
831-1100

40 M 1902 66 ORS
NEIGHBOR, GAYLORD P, 1420 SOUTH 42ND, 66106
831-1100

13 M 1902 41 FP
NEIS, PAUL R, 1929 FEDERAL, 66103

56 M 1902 82 OTO
NELSON, BRENDIA S, 4440 EATON, 66103

57 F 1902 86
NELSON, NANCY A, 3736 CAMBRIDGE, 66103

61 F 1902 87
NEWLIN, PHILIP L, 391S 800TH #6, 66103

61 M 1902 87
NIELSEN, LARRY WAYNE, 15S S 18TH, 66102

371-2330
48 M 2803 75 OBG
NIGH, STEPHEN S, 3900 800TH APT 8, 66103

61 M 1902 87
NOBLE, MARK J, KU MED CENTER-UROLOGY, 66103

588-6148
49 M 2501 75 U
NORRIS, CHARLEY W, KUMC, 66103

588-6700
33 M 1902 64 OTO
NOTHNAGEL, ARNOLD F, 501 WESTVALE, 66102

1S M 1902 39 OO
O'DOYNIK, II, PAUL LEONARD, KU MED CENTER, 66103

588-5000
48 M 1902 73 NS

O'DELL, MICHAEL L, KU MED CTR DEPT OF FP, 66103
588-1908

51 M 1902 77 FP
OTHMER, EKKENHARD, K U MEDICAL CENTER, 66103

588-6440
33 M 40721 66 D
OVERFIELD, A SCOTT, 3838 RAINBOW #906, 66103

57 M 1902
OXLER JR, JOHN EDWARD, 1SS S 18TH, 66103

352-6161
46 M 1902 72 IM

PALMER, MARVIN M, 8919 PARALLEL PKW, 66112
788-9797

45 M 702 71 OBG
PAROO, MANUEL P, K U MEDICAL CENTER, 66103

588-6464
35 M 74801 62 P
PAREKH, AJITKUMAR M, 6013 LEAVENWORTH RD, 66104

299-2069
47 M 49501 71 >UD
PARK, CHAN H, K U MEDICAL CENTER, 66103

588-6029
36 M 58302 62 IM
PARKER, JULIE A J, 4121 THOMPSON APT 8, 66103

61 F 1902 87
PARRA, DANIEL C, 6013 LEAVENWORTH RD, 66104

299-2069
43 M 84703 75 FM
PARRA, MIGUEL O, 6013 LEAVENWORTH RD, 66104

299-2088
37 M 84710 64 FP
PARSONS, JULIE A, 3942 ADAMS #9, 66103

61 F 1902 87
PAVELONIS, JOEL D, 3029 PUCKETT RD APT 27, 66103

57 M 1902 87
PAZELL, JOHN A, 4631 ORVILLE, 66102

287-6464
40 M 2501 66 ORS
PEARSON, MARK A, 4410 RAINBOW, 66103

55 M 1902 87
PECANA, MANUEL C, 8ETHANY MED CENTER, 66102

281-8896
45 M 74801 69 ANES
PENNER, TIMOTHY M, 3700 CAMBRIDGE, 66103

59 M 1902
PERRY JR, LAWRENCE L, KUMC 39TH AT RAINBOW, 66103

588-6522
34 M 1902 59 FP
PERSONS, DIANE L, 1002 W 78TH, 66114

52 F 1902 87
PIERCE, GEORGE E, KUMC - PO BOX 25S, 66103

588-611S
33 M 2307 60 TS
PINGETON, SUSAN K, KU MED CENTER DEPT OF MED, 66103

588-6044
46 F 1902 72 IM
PIPPIN, LYNNE K, KU MED CENTER ANES, 66103

588-6670
45 F 35207 72 ANES
PISCHKE, FRANK J, 1SS S 18TH, 66102

321-1161
35 M 1902 62 OTO
POOREBARAC, FRANCIS A, 3909 800TH APT 4, 66103

59 M 1902 86
PORTER, DAVID M, 4517 TROUP, 66102

287-8800
39 M 4707 64 PD
PORTER, SUSAN S, KU MED CTR ANES DEPT, 66103

588-6670
54 F 1902 79 ANES
PORTER, TAYLOR L, 271S W 42ND APT 11, 66103

60 M 1902 87
POWERS, G ROBERT, 8919 PARALLEL PKWY, 66112

299-1469
33 M 1902 65 FP
PREMSINGH, VALINI G, 4631 ORVILLE #202, 66102

596-2000
39 F 49508 65 CO
PRESKORN, SHELTON H, K U MED CENTER, 66103

588-6400
48 M 1902 74 P
PRESTON, DAVID F, KU MED CENTER, 66103

588-6810
33 M 3841 59 NM
PRETZ, JAMES B, 1610 WASHINGTON BOULEVARD, 66102

342-2442
24 M 1902 47 FP
PRICE, HILTON I, KU MED CENTER - RAD DEPT, 66103

588-6831
49 M 83601 72 OR
PRICE, JAMES GORDON, K U MED CENTER, 66103

588-6510
26 M 702 47 FP
PRIETO, JORGE N, 6013 LEAVENWORTH RD, 66104

299-2069
45 M 26401 69 GS

PROUD, G ONEIL, KU MEDICAL CENTER, 66103

583-6700

13 M 2802 39 OTO

PUCKETT, MICHAEL L. 1851-8 S 31ST, 66106

60 M 1902 87

PUGH, DAVID M. K U MED CENTER, 66103

588-6015

29 M 801 58 CD

QUINN, CHARLES E. 4601 DRVILLE, 66102

287-6604

43 M 4707 68 DBG

RAMZY, MERIT S. 3749 800TH #8, 66103

56 F 1902 86

RANDOLPH III, RICHARD J. 3204 W 43RD, 66103

54 M 1902 87

RECKLING, FREDERICK W. KUMC, 66103

588-6129

34 M 3545 59 ORS

REDDY, EASHWER K. K U MED CENTER - RAD DEPT, 66103

588-7350

44 M 49597 68 TR

REDFORD, JOHN W B. K U MED CENTER, 66103

589-6777

28 M 6501 53 PM

REEB, RONALD JOSEPH, 155 S 18TH, 66102

371-4343

46 M 3006 72 DR

REED, JAMES STEWART, K U MED CENTER, 66103

588-6019

46 M 3601 72 GE

REED JR, WILLIAM D. 155 S 18TH, 66102

371-6805

50 M 2803 77 ORS

REEDER, STEPHEN M. 3127 EATON, 66103

61 M 1902 87

REISWIG, JEFFREY SCOTT, 3808 BODTH #5, 66103

60 M 1902 86

RETHORST, RICHARD D. 4120 THOMPSON #20, 66103

61 M 1902 87

REUSSER, LAYNE M. 3907 ADAMS, 66103

60 M 1902 86

REZAET, SHIRLEY J. STUDENT UNION RDM 276, 66103

58 F 1902 87

RHODAS, DANIEL D. 3101 S SEVENTH, 66103

50 M 1902

RHODAS, JEFFREY P. 2311 MARTY APT #1, 66103

56 M 1902 84

RHODES, JAMES B. KU MEDICAL CENTER, 66103

588-6019

28 M 1902 58 GE

RICE JR, FREDERICK A. 1029 N 32ND ST, 66102

281-5252

36 M 4802 63 JRS

RICH, GARY L. 3014 W 42ND #14, 66206

55 M 1902 87

RICHARDSON, CATHERINE A. 2400 W 38TH #4, 66103

49 F 1902 86

RICHARDSON, JAY L. 8919 PARALLEL, 66112

299-8000

38 M 1902 65 GS

RIGGS, SANDRA L. 3838 RAINBOW #1003, 66103

58 F 1902 87

RILEY, RAY B. 2020 DRVILLE, 66102

06 M 1902 36 DD

RISING, JESSE D. KU MEDICAL CENTER, 66103

588-1934

14 M 1902 38 IM

ROBINSON, DAVID B. KU MEDICAL CENTER, 66103

588-6136

14 M 4101 38 PS

ROBINSON, RALPH G. KU MEDICAL CENTER, 66103

588-6810

37 M 1902 62 NM

ROME, MICHAEL P. 3815 BODTH, 66103

60 M 1902 87

ROOK, LEE E. 4116 STRONG, 66106

831-2834

09 M 1902 38 FP

ROSENTHAL, STANTON J. K U MED CENTER, 66103

588-6800

46 M 1902 71 DR

ROTH, ALAN E. BETHANY HOSPITAL 51 N 12, 66102

281-8814

35 M 1902 62 PATH

RUBIN JR, BEN. 132 S SEVENTEENTH, 66102

371-2561

37 M 3005 61 PD

RUSSO, LIBBIE J. KU MED CENTER, 66103

588-6326

53 F 2846 77 PDE

RUTH, WILLIAM E. K U MED CENTER, 66103

588-6044

26 M 1902 53 PUD

RYAN, MICHAEL J. 764 NEW BROTHERHOOD BLDG, 66101

342-7070

14 M 2834 37 OTJ

SACK, JOSEPH M. 3838 RAINBOW #1204, 66103

60 M 1902 87

SALMON, JAMES S. 2525 W 38TH APT 2D, 66103

56 M 1902 85

SAVIN, VIRGINIA J. KU MED CENTER, 66103

588-6983

44 F 4112 70 IM

SCHIMKE, R NEIL, KU MED CENTER, 66103

588-6043

35 M 1902 62 IM

SCHDELING, RICK D. 2836 EATON, 66103

59 M 1902 86

SCHOTLAND, EDWARD S. 1300 N 78TH STE 303, 66112

334-5250

36 M 86901 66 J

SCHUPP, ELIZABETH A. 301 N 70TH, 66112

57 F 1902 87

SCHUYLER, GREGG T. 5252 SPEAKER RD, 66106

58 M 1902 87

SCHWEGLER, RAYMOND A. 8919 PARALLEL PKWY, 66112

492-6200

37 M 1902 63 CD

SCHWORM, CURTIS P. 155 SOUTH 18TH, 66102

371-4343

47 M 3005 73 DR

SCOTT, JEFFREY, 3731 EATON, 66103

58 M 1902 84

SEIFERT, EARNEST D. K U MED CENTER STUDENT, 66103

56 M 1902 87

SERERES, EDGAR P. 733 N 75TH TERR, 66112

299-9010

15 M 1902 39 FP

SHAW, PAMELA K. 3909 BODTH #11, 66103

60 F 1902 86

SHEEHAN, MAUREEN M. STUDENT UNION BLDG #277, 66103

50 F 1902 86

SHERBON, MARY LOU, 4132 BODTH, 66103

47 F 1902 87

SHERMAN, ROBERT P. 9201 PARALLEL AVE, 66112

334-0040

34 M 1902 63 PUD

SHUMARD, CRAIG J. 3218 EATON, 66103

60 M 1902 86

SIEG, KARL G. 3909 BODTH APT 4, 66103

61 M 1902 86

SIFERS, EARL C. 155 S 18TH, 66102

371-2900

24 M 1902 47 GS

SIFERS, TIMOTHY M. 155 S 18TH, 66102

371-2900

48 M 1902 74 GS

SIMON, JOYCE L. KU MED CTR DEPT OF FP, 66103

588-1910

55 F 1902 79 FP

SINDNY, MARTHA A. 3640 SPRINGFIELD, 66103

58 F 1902 87

SKIKNE, BARRY S. KU MED CENTER, 66103

588-6031

45 M 83601 61 HEM

SNIDER, BRUCE B. 2559 W 46TH, 66103

59 M 1902 86

SNODGRASS, WAYNE R. KU MED CENTER PED DEPT.66103
588-6318
45 M 1720 68 PD
SOMMERFELD, DAVID L. 4455 EATON.66103
-
60 M 1902 86
SOUCEK, CHARLES O. 155 S 18TH.66102
371-4343
31 M 3005 56 R
SOUTHERN, FREDRICK N. 3838 RAINBOW APT 103.66103
-
50 M 1902 87
SPEER, LELAND. 910 N WASHINGTON.66102
-
12 M 1902 36 DD
SPRINGER, MARK J. 2520 W 39TH APT 26.66103
-
61 M 1902 87
STECHSCHULTE, DANIEL J. K U MED CENTER.66103
588-6008
36 M 2834 62 A
STEELE, CLARENCE H. 255 BROTHERHOOD BLDG.66101
321-1161
14 M 1902 40 DTD
STEINZEIG, SHERMAN M. 155 S 18TH.66102
621-1151
25 M 1902 52 CD
STEPHENS, RONALD L. KUMC.66103
588-6029
39 M 1902 65 IM
STEWART, DANIEL L. 3902 BOOTH #3.66103
-
51 M 1902 87
STRAIN, LORRAINE L. 3006 W 46TH.66103
-
58 F 1902 87
STUART, SCOTT P. 3911 ADAMS.66103
-
61 M 1902 87
STUBBLEFIELD, CHARLES T. 155 S 18TH.66102
371-2330
32 M 1902 58 DBG
TARDNER, AMY C. 3915 BOOTH APT 7.66103
-
58 F 1902 85
TAYLOR, ONEITA F. 1863 S 31ST.66106
-
46 F 1902 81 RT
TAYLOR, SARAH A. KU MED CENTER-STUDENT UN.66103
588-6029
50 F 1902 75 IM
TAYLOR, WILLIAM F. 8919 PARALLEL PKWY.66112
299-8000
53 M 2846 76 IM
TEMPLETON, ARCH W. K U MEDICAL CENTER.66103
588-6805
32 M 3005 57 R
THEDINGER, BRADLEY S. KU MED CTR DEPT OF DTD.66103
588-6717
53 M 1902 76 NDTD
THELLMAN, SCOTT T. 4171 CAMBRIDGE.66103
-
58 M 1902 86
THEROU, LEDNA F. K U MED CENTER PED DEPT.66103
588-5908
41 F 6701 67 PD
THOMAS, JAMES H. K U MED CENTER.66103
588-5901
41 M 2012 66 GS
THOMAS, THOMAS V. 211 INDIAN SPRINGS MED BL.66102
287-2600
37 M 49549 61 COS
THOMPSON, DANNIE M. TWO GATEWAY CTR SUITE 917.66101
321-3355
35 M 4707 64 DBG
THORNTON, CAROLYN SUE. 3128 EATON.66103
-
59 F 1902 86
TIOJANCO, REYNA DO R. 6013 LEAVENWORTH RD.66104
799-2069
44 M FP
TORREY, ELIZABETH A. 3901 BOOTH #5.66103
-
60 F 1902 87
TOSONE, STEVEN R. K U MED CENTER.66103
588-6670
52 M 1902 77 ANES
TRUEWORTHY, ROBERT C. KU MED CENTER.66103
588-6340
40 M 2802 66 PD
TURNER, ROBERT N. 3636 SPRINGFIELD.66103
-
59 M 1902 87

VAN DOREN, BRYAN A. 3820 BOOTH APT #10.66103
-
60 M 1902 86
VAN THULLENAR, PHILIP A. BETHANY MED CENTER.66102
281-8834
31 M 2834 57 PATH
VARGHESE, GEDRGE, K U MEDICAL CENTER.66103
488-6798
44 M 49509 69 PM
VATS, TRISHAWAN S. K U MED CENTER PED DEPT.66103
588-6340
40 M 49529 63 PD
VELAROE, HUGO. 4601 ORVILLE AVE STE 14.66102
287-8400
-
M 64 GS
WADDELL, BILL D. 155 SOUTH 18TH.66102
321-0386
31 M 3901 56 IM
WALASZEK, SHEILA J. 3718 STATE LINE.66103
-
59 F 1902 85
WALKER, ANDY E. 3617 CAMBRIDGE.66103
-
61 M 1902 87
WALKER, JACK D. KU MED CENTER.66103
588-1900
22 M 1902 53 FP
WALKER, MAURICE A. 3214 STRONG AVE.66106
831-1433
04 M 1601 28 GS
WALSH, DAVID J. KU MED CENTER PED DEPT.66103
588-6371
46 M 4501 73 PD
WALTER, DONNAL C. KU MED CENTER.66103
588-6337
48 M 1902 76 PD
WALTERS, WILLIAM DAVID. 1428 S 32ND.66106
384-1630
50 M 1902 78 FP
WAMSLEY, CRAIG A. 3733 EATON.66103
-
58 M 1902 86
WEIGEL, JOHN W. UROLOGY DEPT KU MED CTR.66103
588-6148
29 M 1902 54 U
WEINMEISTER, DONALD D. 3733 EATON.66103
-
55 M 1902 86
WEINSTEIN, GARY L. 3824 BOOTH #9.66103
677-1563
-
M 1902
WELLER, ELIZABETH B. KU MED CENTER.66103
588-6464
49 F 60501 75 CHP
WELLER, RONALD ALAN. KU MED CENTER - PSY DEPT.66103
588-6464
48 M 2802 74 P
WETZEL, MARK D. 4172 CAMBRIDGE.66103
-
59 M 1902 86
WHITCOMB, RANDALL W. 4141 ADAMS.66103
-
54 M 1902 81 IM
WHITFIELD, STEVE S. 3806 STATE LINE.66103
-
56 M 1902 82 IM
WIENS, JONATHAN G. 4117 ADAMS APT 213.66103
-
50 M 1902 86
WIENS, LYNN A. 3838 RAINBOW.66103
-
M 1902 87
WILEY, DANIEL M. 2407 W 45TH.66103
-
60 M 1902 87
WILEY, THOMAS M. 3746 STATE LINE APT #1.66103
-
59 M 1902
WILLIAMS, FENTON A. 701 WASHINGTON.66101
281-0361
40 M
WILLIAMS, NANCY J. 3816 STATE LINE.66103
-
60 F 1902 87
WILLIAMS JR, STERLING B. K U MEDICAL CENTER.66103
588-6200
41 M 401 73 DBG
WILSON, LDRI J. 3824 BOOTH APT 7.66103
-
60 F 1902 87
WILSON, PHILIP K. 3600 RAINBOW APT 114.66103
-
61 M 1902 87

WILSON, W TAD, 3728 CAMBRIDGE, 66103

59 M 1902 85
WISE 111, JOSEPH EDWARD, 132 S 17TH, 66102371-2561
51 M 1902 76 PD
WISNER JR, JOHN HENRY, 229 S EIGHTH, 66101588-6431
45 M 1902 76 P
WOLF, KARL T, 621 NORTHRUP AVE, 6610114 M 1902 48 OD
WONDER 11, DONALD C, 2904 W 46TH, 6610353 M 4802 79 GS
WOODROOF, JANET M, 4166 CAMBRIDGE, 6610356 U 1902 87
WRIGHT 111, ROBERT W, 4606 CAMBRIDGE, 66103588-6670
50 M 1902 79 ANES
WRIGHT JR, ROBERT W, 669 NEW BROTHERHOOD BLVD, 66101371-5344
24 M 1902 48 GS
YAKAR, DANIEL, KU MED CTR RAD DEPT, 66103588-3600
32 M 55001 58 RT
ZAREMSKI, SHERMAN C, 4631 ORVILLE STE 209, 66102596-1185
33 M 1720 58 1M
ZIEGLER, DEWEY K, KU MED CENTER, 66103588-6985
20 M 2401 45 N
ZIMBELMAN, ROD O, 3909 800TH APT 12, 6610361 M 1902 87
ZINN, THOMAS W, 155 S 18TH, 66102371-4343
41 M 1902 67 R

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ADAMS, JOYCE A, 6106 HARRISON, 64110

588-6325
50 F 1902 77 PD

ALLEY, ROBYN R, 229 E 46TH APT 3C, 64112

61 F 1902 87
ANDERSON, WILLIAM A, 629 WEST 70TH, 64113281-8881
50 M 2846 76 EM
BENNETT, CHARLES A, 5619 NW HILLSIDE DR, 6415196 M 1902 25 OD
BLACKBURN, TIMOTHY L, 4017 BELL, 6411150 M 1902 86
BRIDGENS, JAMES G, 1025 HUNTINGTON RD, 6411322 M 1902 47 PATH
CHRISTENSEN, SHANE R, A4812 HEINTZ, 64133281-8881
55 M 1902 79 EM
CHRYSANT, STEVEN G, VA HOSP, 64128861-4700
34 M 41801 59 CO
CLAY, MICHAEL J, 1717 1/2 WESTPORT RD, 6411158 M 1902 87
COLLINS, DAVID E, 3700 PENNSYLVANIA, 6411158 M 1902 87
COLVER, JEFFREY W, THE REGENT SUITE 36, 6411250 M 1902 86
CRAIG, JEREMIAH E, 3630 BELL, 64111531-6001
54 M 1902 79 1M
DE JONG, JOHN THEODORE, 3716 BELL, 6411159 M 1902 86
DIEHL, ANTONI M, 4320 WORNALL RD, 64111753-4414
24 M 2604 47 POC
DO, SON T, 1301 VALENTINE RD APT 4, 6411161 M 1902 86
FAIRMAN, DAN S, 1808 W 41ST APT 2E, 64111

55 M 2834 82 1M

FALLON, JOHN H, 3730 WYOMING, 64111

59 M 1902
FESTOFF, BARRY W, VA HOSPITAL, 64128861-4700
40 M 1102 77 M
FRECHETTE, ALAN R, 119 N LAWN, 6412357 M 1902 87
GLATTER, THOMAS R, VA HOSPITAL, 64128861-4700
34 M 1611 60 1M
GOODFREY, ROBERT G, VA HOSP, 64128861-4700
27 M 1902 58 RHU
GOLOSTEIN, ALAN O, 901 W 113 TERRACE, 6411461 M 1902 87
HAGMAN, JENNIFER O, 3560 WYOMING, 6411160 F 1902 86
HAMILTON, WILLIAM A, 3730 WYOMING, 6411159 M 1902 86
HARD, BENJAMIN F, 8400 HAWTHORN RD, 64120242-2574
28 M 4802 55 OBG
HAYES, DAVID M, 3729 STATE LINE, 6411155 M 1902 85
HOMLY, EVE K, 3923 HARRISON, 6411040 F 1902
HUGHES, STEVEN, 4326 WYOMING, 6411145 M 1902 87
HYMAN, HENRY T, O.O., 5500 N OAK SUITE 201, 6411849 M 2879 76 OBG
JOHNSON, FREDERICK E, C/O MARY K BLOTT, 6411292 M 2843 21 OD
JOHNSON, TERESA F, 4122 BELL, 6411155 F 1902 81 GS
JOHNSTON, JAN M, 804 W 48TH APT 203, 6411257 F 1902 83 1M
KINPORTS JR, EDWARD B, 2400 PERSHING RD STE 561, 64108842-1950
46 M 3006 74 EM
KINPORTS SR, EDWARD B, 2520 GRAND AVE #309, 6410815 M 1602 42 EM
LESSIN, DIANNA L, 3706 WYOMING #10, 6411155 F 1902 87
LEWIS JR, H DANIEL, VETERANS MEDICAL CENTER, 64128861-4700
32 M 5101 58 CO
LICHTENHAN, JOHN B, 1135 W 41ST S STUDIO, 6411161 M 1902 87
LILLICH, MAUREEN A, 4804 JARBOE, 6411259 F 1902 86
LUCKEROTH, LEAH L, 4804 JARBOE, 6411258 F 1902 86
LUETJE, CHARLES MARION, 2928 MAIN SUITE 105, 64108561-4423
41 M 2803 67 OTO
MCVEY, PAMELA S, 3217 KARNES BLVD, 64111842-1950
56 F 1902 80 EM
MILLS, KIRK C, 3747 WYOMING APT 3, 6411157 M 1902 86
MILLS, MELISSA J, 1311 W 44TH TERR APT 106, 6411157 F 3605 83 1M
ORR, STEVEN M, 4544 N OLIVE, 6411655 M 1902 79 EM
PAYNE, J RALPH, 4450 ROCKHILL TERRACE, 64110334-2500
40 M 1902 62 EM
PERICO, CARLOS J, 5801 E 113TH, 64134761-1866
32 M 26404 60 FP
PINKHAM, CHRIS M, 5311 HARRISON, 64110

53 M 1902 87

PDSS, WILLIAM B. 4144 HARRISON.64110

61 M 1902 87
PRENDES, CARLOS A. 2900 BALTIMORE SUITE 400.64114
753-5700
50 M 3005 79 GP
REDING, DOUGLAS J. 428 W 60TH TERR.64113
588-5000
54 M 1803 80 IM
RUHL, CONSTANCE E. 3711 GENESSEE.64111

54 F 1902 86
SALEH, GEORGE A. D.O., 5500 N JAK SUITE 201.64118
452-7300
50 M 2878 D8G
SANDERS, JAMES E. 1301 VALENTINE #5.64111

51 M 1902 86
SCOTT, STEVE G. 1310 MANHEI M.64109

51 M 1902 86
SINGER, PHILIP A. V A HOSPITAL.64128
861-4700
42 M 3545 69 M
SLAVIK, MILAN. V A HOSP.64128
588-3302
30 M 28602 60 ON
STASS-ISERN, MERRILL. 1405 W 50TH TERR.64112
281-8880
50 M 84706 77 EM
STOKES, ROBERT LEE. 3621 BELLEVUE.64111
281-8882
43 M 4804 74 EM
STOVE, CHRIS D. 1135 W 41ST APT 5.64111

83 M 1902 87
TEARE, MARIJD. 1804 W 41ST APT 1E.64111

60 F 1902 87
UTLEY, JAMES HARMON. 4951 WESTWOOD TERR.64112
281-8880
51 M 1606 74 EM
VAN BUSKIRK, WILLIAM C. 6700 TROOST SUITE 308.64131
361-1822
23 M 2401 46 GS
VITALE, NEIL B. 1135 W 41ST.64111

55 M 1902
WESSELIUS, LEWIS J. VA HOSPITAL.64128
861-4700
50 M 1902 82 PUO
WIEGMANN, THOMAS B. VETERANS MED CENTER.64128
43 M 40902 69 NEP
WILNER, JAY. 1615 W 39TH.64111
58 M 1902 87

KINGMAN—316 (Ninnescah Society)

BLOOM, L THEIL. 80X 496.67068
32 M 1902 57 R
BOYER, ROBERT E. 760 AVENUE D WEST.67068
532-5145
36 M 1902 63 FP
BURKET JR, GEORGE E. SPRING LAKE RTE 1.67068
12 M 1902 37 OD

KINSLEY—316 (Iroquois County Society)

ATWOOD, M DALE. 616 NILES.67547
659-2114
19 M 1902 51 FP
MCKIM, W LYNN. 109 WEST 8TH.67547
653-2137
33 M 1902 59 FP
SCHNOEBELEN, REVE E. 807 EAST 4TH.67547
659-2141
16 M 3901 40 FP

KIOWA—316 (Tri-County Society)

CHRISTENSEN, MARION D. 220 SOUTH EIGHTH.67070
825-4121
25 M 3901 52 FP

RODRIGUEZ, ALBERTO. 425 CAMPBELL.67070
825-4039
25 M 27501 49 GP

LA CROSSE—913 (Central Kansas Society)

SHARGAVA, ASHOK KUMAR. SHARGAVA CLINIC.67548
222-2564
37 M 49547 64 FP
SHARGAVA, SHOBHANA. SHARGAVA CLINIC.67548
222-2564
38 F 49547 64 FP

LARNED—316 (Pawnee County Society)

CRAM JR, DLE R. 722 MANN.67550
285-2141
18 M 1902 43 FP
DAVIS, DAVID H. 815 W 6TH.67550
04 M 1902 30 00
EWING, THOMAS D. 804 CARRDLL.67550
285-3133
22 M 1902 46 FP
SHAH, MIAN. SHAH CLINIC.67550
285-3173
32 M 70403 58 GS
SHAH, NASREEN. SHAH CLINIC.67550
285-3173
39 F 70409 62 J8G
SHEPARD, LEROY W. 603 W 5TH.67550
04 M 3006 30 00
SMITH, JOHN D. 804 CARROLL.67550
285-3133
22 M 3901 51 FP
WAGNER, LENARD D. 923 CARROLL.67550
54 M 512 79 FP

LAWRENCE—913 (Douglas County Society)

BAILEY, WILLIAM A. PO BOX 127.66044
843-9125
40 M 1902 66 ORS
BELOT JR, MONTI L. LAWRENCE NATIONAL BK BLDG.66044
843-3640
13 M 1902 40 FP
BIERI, PETER V. 1112 W SIXTH ST.66044
841-5217
45 M 1902 71 ENT
BISHOP, RODNEY LEE. LAWRENCE IM ASSOC.66044
842-7200
49 M 1902 75 IM
BITTENBENDER, LEE R. 930 IOWA.66044
842-7001
46 M 1902 72 O
BOYDEN, MARY S. MEDICAL ARTS BLDG.66044
842-3778
14 F 2604 38 POA
BRANSON, VERNON L. 346 MAINE.66044
842-4477
17 M 1902 42 PO
BRUNFELDT, J DAN KRAUS. 404 MAIN.66044
842-3635
52 F 1902 77 IM
BUCK JR, HENRY #. 1112 W SIXTH.66044
841-9200
34 M 1902 60 OBG
CHEDIAK, ELIAS. 601 MISSOURI.66044
841-7430
39 M 84704 65 P
CLINTON, DALE L. 15 E SEVENTH SUITE 103.66044
841-5716
21 M 1902 54 GP
CULVER, WARREN T. 2500 W 6TH.66044
842-4178
20 M 3508 46 OPH
DUNLAP, RICHARD L. MEDICAL ARTS CENTER.66044
842-4344
12 M 3005 37 EENT
FRIESEN, DALE. PO BOX 521.66044
842-7026
47 M 1902 74 ANES

GILLES, HELEN M. 1301 IOWA.66044

22 F 1902 45 DO
GODWIN, PHILLIP A. 500 ROCKLEDGE.66044841-6540
28 M 1902 55 ANES

GRAY, C K. 3310 CLINTON.66044

842-7200
48 M 1902 75 IM

GRAY, SCOTT E. 346 MAINE.66044

843-0677
53 M 1902 79 DBG

HAGGAN, MARGARET E. 1746 N H.66044

F 2501 42 DO

HASSELLE III, JAMES E. 601 MISSOURI.66044

841-7430
35 M 4706 59 P

HATTON, DONALD W. 404 MAINE ST.66044

842-3635
42 M 1902 68 IM

HERMES, RICHARD L. THE MEDICAL ARTS CENTER.66044

843-0677
15 M 4112 39 DBG

HIEBERT, DAVID L. 1112 W SIXTH.66044

841-3211
36 M 1902 61 R

HIRD, WAYNE E. 2802 TRAIL RD.66044

842-4300
26 M 1902 50 TS

HUGHES, ROBERT WALTER. MEDICAL ARTS BLDG.66044

843-1374
27 M 1902 54 FP

INGHAM JR, H LAIRD. 404 MAINE.66044

842-3635
45 M 3901 70 IM

JONES, H PENFIELD. MED ARTS CENTER.66044

06 M 2401 31 GS

JOSEPH, HOWARD F. DOCTORS BUILDING.66044

843-3981
26 M 1902 51 U

KEELER, LINDA L. MTL HLTH CL WATKINS MEM.66045

47 F 1902 76 P

LEARNED, GEORGE R. 401 ARKANSAS.66044

843-5502
22 M 1902 55 GS

LESSENDEN, GLENN A. 1112 W SIXTH SUITE 110.66044

843-6233
24 M 1902 48 FP

LOVELAND, G CHARLES. 346 MAINE.66044

842-4477
47 M 1902 73 PD

MAUSEN, GLENN L. 1112 W SIXTH.66044

841-3211
38 M 3005 65 R

MAGEE, LAWRENCE M. 233 DAKOTA.66044

325-2240
52 M 1902 77 FP

MANAHAN, G EUGENE. MED ARTS CENTER.66044

842-0211
19 M 1902 44 GS

MITCHELL, ALEX C. 1626 W 20TH.66044

843-4739
19 M 1902 50 PH

MODDRELL, CAROL A. 404 MAINE.66044

749-6100
45 F 1902 71 PATH

MONCKTON, LAJRANCE A. 1112 W SIXTH SUITE 204.66044

843-2010
48 M 1902 74 GS

MYRICK, STEPHEN W. 346 MAINE.66044

842-6644
52 M 1902 77 GS

NELSON, RICHARD D. 935 IOWA.66044

843-0921
11 M 1001 41 FP

DELSCHLAGER, RONALD D. 1112 W SIXTH.66044

841-3211
43 M 1902 69 R

OLSON, CARL E. 935 IOWA.66044

842-9911
17 M 1611 46 FP

ORCHARD, RICHARD A. 1112 W SIXTH SUITE 202.66044

841-2280
41 M 2802 68 DPH

OSBERN, LIDA. 404 MAINE.66044

842-3635
52 F 1902 77 IM

OWEN, GARRY D. 1112 W SIXTH.66044

841-9200
37 M 1902 63 DBG

PEES JR, GERALD BOYD. 1112 W SIXTH SUITE 210.66044

843-5160
45 M 1902 71 IM

PLACEK, DEBRA C. MED ARTS CENTER.66044

843-0677
53 F 3005 78 DBG

PRAEGER, MARK A. 1112 W SIXTH SUITE 204.66044

843-2010
42 M 1902 68 GS

REED, JAMES S. WATKINS MEMORIAL HOSP.66045

843-4455
23 M 1902 47 FP

REED, RALPH R. 404 MAINE.66044

842-3635
27 M 1902 53 IM

REESE, JOHN L. 346 MAINE.66044

842-6644
35 M 1902 61 GS

REITH, PAUL. 1331 NAISMITH.66044

M 2834 IM

RIORDAN, TERRANCE. 346 MAINE.66044

842-4477
51 M 1902 77 ADL

ROBERTS, RICHARD S. 308 MAINE.66044

843-6137
17 M 2802 44 GS

SANDERS, J ALAN. LAWRENCE MEMORIAL HOSP.66044

842-2083
29 M 1902 60 PATH

SCHMOSE, GREGORY D. 1112 W SIXTH SUITE 210.66044

843-5160
51 M 1902 76 IM

SCHROEDER, SYDNEY D. 1112 W SIXTH SUITE 112.66044

841-4311
18 M 1902 44 P

SCHWEGLER, RAYMOND A. 1504 UNIVERSITY DR.66044

07 M 2604 31 DO

SEGEBRECHT, STEPHEN L. 3012 OXFORD RD.66044

841-5217
55 M DTD

SOSINSKI, RICHARD F. 1112 W SIXTH.66044

843-5160
51 M 1902 76 IM

STIVERS, MARK T. 404 MAINE.66044

842-2083
47 M 2803 73 PATH

TUCKER, DONALD R. 1517 INDIAN WELLS CT.66044

354-5275
31 M 1902 57 IM

WELL, MICHAEL A. 45 COUNTRY CLUB TERR.66044

749-0639
41 M 1606 67 U

WERTZBERGER, JOHN. PO BOX 127.66044

843-9125
36 M 1902 63 ORS

WERTZBERGER, KENNETH LYNN. 1112 W SIXTH BOX 127.66044

843-9125
47 M 1902 73 ORS

WILCOX SR. HOWARD L. MEDICAL ARTS CENTER.66044

843-0677
19 M 3520 44 DBG

WOLLMANN, MARTIN. 2615 ORCHARD LN.66044

843-4455
26 M 1902 57 IMLEAVENWORTH—913
(Leavenworth County Society)

AL-BAGHAL, MOHAMMAD. 113 DELAWARE SUITE A.66048

651-2200
45 M 87501 72 U

ANWAR, M ZIA. 109 DELAWARE.66048

651-2977
39 M 11801 67 IM

ASHKAR, ADNAN A. 109 DELAWARE SUITE F.66048

682-6818
42 M 52801 73 DBG

BARRY, DAVID R. 500 EISENHOWER RD.66048

727-6000
42 M 1902 68 FP

BUESING, OLIVER R. 230 NINTH.66048

10 M 2501 32 GS

CENAC, MARK T. 500 EISENHOWER RD.66048

727-6000
27 M 401 49 GS

COMBS, PETER S. 213 DELAWARE.66048

682-0242
14 M 4101 41 IM

DE SOUZA, DERRICK J. 2201 S FOURTH.66048
651-6030
43 M 49501 66 GS
DECENA, IMMACULADA, PO BOX 1675 VAMC.66048

45 F 74810 68 EM
DUYSAK, SAMI. 520 6TH AVE.66048
682-6661
22 M 90201 47 IM
FEVURLY, CHRIS D. 500 EISENHOWER.66048
727-6000
54 M 1902 79 IM
GERBER, HARRY A. 605 N SIXTH.66048

96 M 5606 30 FP
GRAHAM, KENNETH L. RTE 2 BOX 182AA.66048
727-6000
21 M 3840 45 GS
GRAHAM, THOMAS W. 500 EISENHOWER RD.66048
727-6000
26 M 3840 50 IM
GRISOLIA, ANDRES. 424 WALNUT ST.66048
682-5400
27 M 84708 50 ORS
HAMMEKE, JOHN C. 3601 S 4TH ST TRAFFICWAY.66048
682-5201
27 M 401 61 DPH
JOHLER, TERRY HARTWIG. 500 EISENHOWER RD.66048
727-6000
40 M 2802 67 FP
JOHNSON, PAUL D. 520 SIXTH AVE.66048
682-6661
36 M 1902 61 FP
KAVI, NAGESH G. 3116 LAKEVIEW CL.66048
682-2000
32 M 49509 58 IM
LIPOFF, JAY I. 410 S FOURTH.66048
682-6950
50 M 3519 75 IM
LOHRENTZ, LOIS H. 113 DELAWARE STE I.66048
682-1189
26 F 1902 52 ANES

MCCOLLUM, WILLIAM B. 3601 S FOURTH.66048
682-6661
41 M 1902 66 TS
MERRITT, W HENRY. 44 WESTWOOD DR.66048
682-6661
14 M 702 39 GS
MILLS, VERNON A. 4510 S 4TH TRFWAY.66048
682-6661
51 M 1902 77 PD
PARKER, ROBERT W. 500 EISENHOWER RD.66048
727-6000
45 M 1902 71 FP
PRAY, CLAUDIA M. 529 DELAWARE.66048
682-4771
52 F 1902 74 PD
RABE, MELVIN A. 600 S BROADWAY.66048

14 M 1902 37 DD
SNOW, DONALD L. MED ARTS BLDG.66048
682-1000
21 M 64901 54 DBG
STRUTZ, WILLIAM C. 68 WESTWOOD DR.66048
682-8868
39 M 5606 43 R
VOORHEES, CARROLL D. 520 SIXTH AVE.66048
682-6661
25 M 1902 52 FP
VOORHEES, GORDON S. 520 SIXTH AVE.66048
642-6661
12 M 1902 39 IM
WALTZ, CHARLES A. 520 SIXTH AVE.66048
682-6661
35 M 3840 62 TS
WASHBURN, MICHAEL E. 4516-B S 4TH TRAFFICWAY.66048
727-2322
47 M 4705 73 GS

LEBO—316
(Flint Hills Society)

HUNTER, KENNETH R. .66856
256-2565
07 M 1902 39 FP

LENORA—913
(Northwest Kansas Society)

STEICHEN, EDWARD F. .67645
05 M 1601 31 FP

LIBERAL—316
(Seward County Society)

ALLEN, RAY E. 2 PLAZA DR.67901
624-5691
37 M 1902 64 IM
CAEDO, CARMELITA D. PO BOX 1643.67901
624-1651
41 F 74801 63 R
CAMPION, WOODROW M. 121 W THIRD.67901
624-2594
13 M 1902 39 IM
ESTRADA, EDMUNDO C. 1023 N KANSAS SUITE 3.67901
733-1331
43 M 74801 67 GS
ESTRADA, LINA. 1023 N KANSAS SUITE 3.67901
733-1331
43 F 74801 68 PD
GRIMES, I ROSS. 222 W 15TH.67901
624-1676
27 M 3901 54 TS
HARRIS, NORVAN D. PO BOX 1069.67901
624-3811
20 M 1902 44 DBG
HOLCOMB, WILLIAM M. 15 E 11TH.67901
624-2252
31 M 3901 56 GS
KOONS, JESS W. 1210 N WASHINGTON.67901
624-3841
27 M 1902 57 DPH
NEVINS, RICHARD L. PO BOX 1824.67901
624-1841
47 M 3901 73 FP
PROCHAZKA, OTTO F. BOX 1809.67901
12 M 1902 38 DD
RATHBUN, EDWIN D. 610 W 11TH.67901
624-1841
36 M 1902 62 FP
REESE, JACK D. 15 E 11TH.67901
624-6226
32 M 1902 57 FP
WADE, THEODORE E. 318 N LINCOLN.67901
354-5275
04 M 512 30 DD
ZAINALI, ASSADOLLAH. 601 LILAC DR.67901
524-1651
46 M 51701 72 R

LINDSBORG—913
(McPherson County Society)

FREDRICKSON, DUANE E. 121 W LINCOLN.67456
227-3371
39 M 1902 66 FP
FULLER, DERYL D. RR 2 BOX 6A.67456
227-2747
25 M 1902 50 FP
MURFITT, MALCOLM C. 231 N MAIN.67456
227-2732
13 M 801 41 FP

LYNDON—913
(Franklin County Society)

STOUT, NILES M. .66451
828-4521
16 M 1902 50 FP

LYONS—316
(Rice County Society)

GRIMES, JAMES T. 1221 W NOBLE.67554
257-5124
27 M 1902 53 FP
SIEMENS, RICHARD A. 1221 W NOBLE.67554
257-5124
30 M 1902 59 FP

TOBIAS, ROGER R. 1017 S BELL.67554
278-2123
51 M 1902 76 FP
WOLF, CURTIS V. 1221 W NOBLE.67554
257-5124
37 M 1902 64 FP

MADISON—316
(Flint Hills Society)

BROWNING, WILLIAM R. 205 W MAIN.66860
437-2140
44 M 1902 73 FP
PARKER, WAYNE G. RR #1.66860
437-2907
27 M 1902 56 FP

MANHATTAN—913
(Riley County Society)

BAKER, RICHARD B. 2600 ANDERSON.66502
537-4200
42 M 4113 68 DR5
BALL, RALPH G. 215 S DELAWARE.66502
-
03 M 1902 27 00
BAMBARA, JOHN F. PO BOX 128.66502
539-5363
46 M 1902 75 PATH
BARLOW, JOHN M. 1133 COLLEGE.66502
539-3504
45 M 1102 71 0TD
BASCOM, CHARLES H. C/O LAFENE STU HLTH CTR.66506
532-6544
31 M 1902 55 FP
BASCOM, GEORGE S. 1133 COLLEGE.66502
539-5341
27 M 2401 52 GS
BOESE, KENNETH M. 1133 COLLEGE AVE.66502
776-4744
25 M 1902 56 FP
BROWN, ROBERT M. 1133 COLLEGE.66502
537-4940
31 M 1902 63 FP
BURDICK, BRUCE M. 117 S FIFTH.66502
776-9411
25 M 512 53 P
CATHEY, ROBERT H. 1133 COLLEGE AVE.66502
537-4990
42 M 1902 68 0
CRANE, C HERBERT. 1133 COLLEGE.66502
537-9030
22 M 3520 46 PD
DURKEE, WILLIAM R. 1133 COLLEGE AVE.66502
776-4744
23 M 1902 45 1M
FAIRCHILD, JOHN A. 756 COLLEGE HTS CIR.66502
-
14 M 3006 41 00
FISCHER, REX R. 1133 COLLEGE.66502
776-1400
34 M 3005 60 0BG
FREEMAN, FRED A. MANHATTAN MED CENTER.66502
537-8710
42 M 1902 69 U
GARNER, JAMES O. 1133 COLLEGE AVE.66502
537-4940
43 M 2834 71 1M
HANCOCK, DANIEL E. 1133 COLLEGE PO BOX 128.66502
539-5363
45 M 2803 PATH
HAUN, RUOY T. 1133 COLLEGE.66502
537-3888
49 M 1902 78 0BG
HEASTY, ROBERT G. 2030 SCHEU DR.66502
-
11 M 3519 38 00
HOSTETTER, PHILIP H. 821 POYNTZ.66502
537-2544
17 M 1902 42 FP
JUBELT, MILBERT P. 1133 COLLEGE.66502
537-9030
19 M 1611 43 PD

KALOOR, RICHARD H. PO BOX 128.66502
539-5363
40 M 2401 66 PATH
KLINGLER JR, EUGENE A. 1133 COLLEGE.66502
539-5341
35 M 1902 62 GS
KLOBASA, CHARLES L. 210 SOUTHWIND PLACE.66502
539-5337
49 M 2803 75 CHP
LAFENE, BENJAMIN W. 1844 ANDERSON AVE.66502
-
01 M 3806 31 00
LOVE, STANLEY W. 1133 COLLEGE AVE.66502
776-3451
32 M 1902 59 OPH
LYONS JR, FRANK C. 1133 COLLEGE AVE.66502
539-7641
44 M 3840 70 OR
MARSHALL, RONALD L. 1133 COLLEGE AVE.66502
539-5322
42 M 3005 67 0BG
MARTIN, DANIEL C. LAFENE HEALTH CENTER KSU.66506
532-6544
30 M 1902 58 1M
MCKNIGHT, DAVID E. 1133 COLLEGE AVE.66502
539-7641
32 M 1902 62 R
MCNEIL, ELBERT O. 1133 COLLEGE.66502
537-9030
22 M 702 48 PD
MEEK, PALMER F. 1133 COLLEGE.66502
537-2651
45 M 1902 71 1M
MILLER, ABRAHAM H. 1133 COLLEGE.66502
527-2651
29 M 4101 54 1M
MOSIER, MICHAEL L. 215 SOUTHWIND PL.66502
776-9761
52 M 1902 77 FP
MOSIER, STEVEN J. 215 SOUTHWIND PLACE.66502
776-9761
49 M 1902 74 FP
MOWRY, GERALD L. 1133 COLLEGE.66502
776-1400
26 M 1902 53 0BG
OLNEY, ROBERT O. 1133 COLLEGE AVE.66502
539-7555
27 M 3005 51 GS
PETERSON, JACK T. 1133 COLLEGE.66502
539-5363
25 M 1902 50 PATH
PHILIPP, JOSEPH THEODORE. 1115 WATERS.66502
537-7373
45 M 1902 67 OPH
PHILLIPS, STEPHEN B. 1501 JARVIS.66506
-
17 M 1902 45 00
REITZ, LELAND C. 1133 COLLEGE.66502
537-2651
36 M 1902 63 1M
REITZ, ROGER P. 1133 COLLEGE.66502
537-2651
32 M 1902 59 1M
ROSE, GRAHAM C. 1133 COLLEGE.66502
537-9030
46 M 4706 70 PD
SHIELDS, THOMAS M. 1133 COLLEGE.66502
539-5341
49 M 1902 74 GPVS
SNYDER, KELVIN K. 1133 COLLEGE.66502
537-9030
52 M 1902 80 PD
STONE, G REX, RT 1, BOX 182.66502
29 M 1902 54 GS
TAYLOR, BARBARA O. 1133 COLLEGE.66502
357-4940
50 F 1902 75 1M
TIEMANN, WILLIAM H. 1133 COLLEGE.66502
537-4940
42 M 3005 67 FP
TOUT, ROBERT C. LAFENE STUDENT HLTH CTR.66506
532-6544
27 M 4812 53 FP
VOLKMAN, H. HARLEY W. 1133 COLLEGE AVE.66502
539-7641
47 M 1902 72 R
WALL, KEVIN K. 215 SOUTHWIND P.66502
776-9761
53 M 2101 79 FP
WHITE, THADDEUS H. 1735 ANDERSON AVE.66502
-
15 M 1902 42 00

MANKATO—913
(*Republic County Society*)

KIMBALL, RICHARD R. 102 S CENTER. 66956
378-3511
45 M 1001 72 FP

MAPLE HILL — 913
(*Flint Hills Society*)

KEITH, ROBERT MARSHALL, RURAL ROUTE 1. 66507
27 M 801 54 P

MARYSVILLE—913
(*Northeast Kansas Society*)

ARGD, DONALD A. 808 N 19TH. 66508
562-2303
36 M 300S 64 FP
LAWS, LEWIS R. 808 N 19TH. 66508
562-2303
25 M 1902 54 FP

MAYETTA — 913
(*Shawnee County Society*)

BEATY, JAMES R. RT 2 BOX 11. 66509
295-8090
32 M S101 66 EM

McLOUTH—913
(*Shawnee County Society*)

SNOOK, ROBERT RUFUS. 66054
796-6116
11 M 1902 42 FP

McPHERSON—316
(*McPherson County Society*)

BAYBROOK, WAYNE KIRK. 915 N WALNUT SUITE 312. 67460
241-3100
47 M 512 78 ORS
BILLINGS, THOMAS. PO BOX 1327. 67460
241-5500
39 M 1902 66 FP
BRANDSTED, ERNEST C. 400 W 4TH. 67460
241-1654
18 M 1606 44 OBG
CLAASSEN, SAMUEL D. 400 W FOURTH. 67460
241-7033
53 M 1902 78 1M
COLLIER, WILLIAM J. 400 W 4TH. 67460
241-1766
25 M 360S 49 TS
DYCK, ARTHUR H. C/O PEOPLES BANK/TRUST. 67460
241-0357
33 M 1902 28 FP
FERREE, RICHARD ALLAN. 400 W FOURTH. 67460
241-7400
51 M 3006 76 FP
FIELDS, GALEN W. 333 C - S LAKESIDE DR. 67460
15 M 1902 49 OD
GILLAN JR, DALE EDWIN. 915 N WALNUT APT 312. 67460
241-7450
53 M 1902 78 GS
JOHNSON, J RICHARD. 400 W 4TH. 67460
241-4293
23 M 1902 55 1M
PIERSON, WEIR. BOX 1028. 67460
241-1445
17 M 1902 44 FP
PRICE, VAUGHAN C. PO BOX 451. 67460
05 M 4706 29 GS
SCHURLE, DALE R. 400 W FOURTH. 67460
241-7033
53 M 1902 78 1M
THOMAS, GREGORY MCQUEEN. 400 W FOURTH. 67460
241-7400
47 M 1902 73 FP

MEADE—316
(*Iroquois County Society*)

FELDMAYER, SEELEY T. PO BOX 1030. 67864
873-5432
46 M 74811 80 GP
HILL, RICHARD H. 234 EAST CARTHAGE. 67864
873-2113
18 M 1902 44 FP

MEDICINE LODGE—316
(*Tri-County Society*)

HOFFER, JOHN G. 910 N WALNUT. 67104
886-3222
13 M 1902 44 GS
STUCKY, DEAN E. 901 N WALNUT. 67104
886-5653
33 M 1902 60 FP

MINNEAPOLIS—913
(*Saline County Society*)

BARKER, STEVEN E. 311 N MILL. 67467
392-2144
51 M 1902 76 FP
WEDEL, KENNETH D. 311 N MILL ST. 67467
392-2144
32 M 1902 60 FP
WEDEL, KERMIT G. 311 N MILL ST. 67467
392-2144
32 M 1902 60 FP

MINNEOLA—316
(*Iroquois County Society*)

STEPHENS, CHARLES. MINNEOLA CLINIC. 67865
885-4202
33 M 2803 58 FP

MONTEZUMA—316
(*Ford County Society*)

CURA, ELSA C. MONTEZUMA CLINIC BOX 384. 67867
846-2251
37 F 74808 61 1M

MOUNDRIDGE—316
(*McPherson County Society*)

KAUFMAN, WILLARD E. 115 N CHRISTIAN AVE. 67107
345-6322
28 M 1902 53 FP
LOGANBILL, VARDEN J. 115 N CHRISTIAN AVE. 67107
345-6322
26 M 1902 54 FP

MULVANE—316
(*Sedgwick County Society*)

CORB, LESLIE H. 102 E MAIN. 67110
777-1441
17 M 4804 47 FP
TURKLE, JANET K. 506 RIVERDALE. 67110
58 F 1902 86

NASHVILLE—316
(*Ninnescah Society*)

WAYLAN, THORNTON L. 67112
06 M 1902 35 OD

NEODESHA—316
(Southeast Kansas Society)

CHRONISTER, BERT, PO BOX 118, 66757
325-2622
38 M 1902 64 FP
MOOREHEAD JR, F ALLEN, 709 MAIN ST, 66757
325-2200
39 M 1902 65 FP

NESS CITY—913
(Central Kansas Society)

PRAKALAPAKORN, DARANE, 412 N TOPEKA, 67560
798-2233
47 F 89101 69 PD
PRAKALAPAKORN, YANYONG, 412 N TOPEKA, 67560
798-2233
43 M 89101 69 GS

NEWTON—316
(Harvey County Society)

ALLEN, FRANCES A, 1112 30YD, 67114
-
15 F 1902 43 DO
BATES, MICHAEL NICHOLS, 215 S PINE SUITE 302, 67114
283-4153
50 M 1902 75 JBG
BENTON, JAY S, 301 MAIN, 67114
283-7257
23 M 4804 49 OBG
BO, T, MICHAEL, AXTELL CLINIC, 67114
283-2800
51 M 1902 76 GS
CAMPBELL, FRANCES S, 1901 E FIRST, 67114
283-2400
35 F 4101 61 P
CARPER, IVAN H, 203 E BROADWAY, 67114
283-2800
28 M 1902 59 GS
CARPER, OWEN E, AXTELL CLINIC, 67114
283-2800
37 M 1902 64 FP
CLAASSEN, MILTON A, 201 S PINE ST, 67114
283-3600
32 M 1902 58 JRS
CRAIG, CHARLES C, AXTELL CLINIC, 67114
283-2800
45 M 1902 71 ORS
DE FREST, DIANE J, 1616 BERRY, 67114
-
60 F 1902 86
DYCK, GEORGE, PRAIRIE VIEW INC, 67114
283-2400
37 M 6201 64 P
ENNS, EUGENE K, 6 INDIAN LANE, 67114
-
15 M 1902 40 DO
FENT, LEE S, 316 OAK, 67114
283-0505
14 M 2834 43 GS
FRANSEN, HERBERT, 215 S PINE, 67114
283-0033
32 M 6501 60 GS
FRANSEN, PAUL M, BOX 848, 67114
283-5040
46 M 6501 71 FP
GLOVER, RICHARD M, AXTELL CLINIC, 67114
283-2800
21 M 1902 53 FP
GRABER, CHARLES, 215 S PINE, 67114
283-0033
44 M 3006 75 GS
GRISWOLD, DALE G, AXTELL CLINIC, 67114
283-2800
27 M 1902 53 IM
GROVE, JOHN A, 407 W 16TH, 67114
-
08 M 1606 37 ORS
HENORICKSON, JON R, AXTELL CLINIC, 67114
283-2800
51 M 1902 73 PD
HENORICKSON, KATHRYN D, AXTELL CLINIC, 67114
283-2800
52 F 1902 77 PD
HWA, EUGENE C, 500 MAIN, 67114
283-1160
21 M 24216 47 R

IRWIN, RICHARD L, 218 S KANSAS AVE, 67114
283-1400
49 M 1902 75 JPH
ISAAC, CHARLES A, 203 E BROADWAY, 67114
283-2800
25 M 1902 49 U
KLIEWER, VERNON L, PRAIRIE VIEW MHC, 67114
283-2400
31 M 1606 57 CP
KUMAR, SURINDER, 201 S PINE, 67114
835-2241
46 M 1902 69 OBG
LINDHOLM, GERALD R, AXTELL CLINIC, 67114
283-2800
51 M 1902 76 FP
NACHTIGALL, ANDREW, BETHEL CLINIC, 67114
283-3600
28 M 1902 59 PD
OLSON, ERWIN T, BETHEL CLINIC, 67114
283-3600
19 M 1902 47 PD
PRENTISS, HAROLD, 1305 TERRACE DR, 67114
283-9433
36 M 1720 62 R
QAMAR, YUSUF, 203 E BROADWAY, 67114
283-2800
38 M 70409 62 IM
RAODVAND, RAOMILA, BOX 364, 67114
-
34 F 95702 60 R
RICH, ELOON S, BETHEL CLINIC, 67114
-
16 M 1902 46 DO
SCHMIDT, HERBERT R, CEDAR VILLAGE, 67114
-
03 M 1902 34 DO
SILLS, CHARLES T, 1631 HILLCREST, 67114
-
09 M 1902 37 DO
SIMMONS, ROBERT EARLE, BOX 848, 67114
283-5040
49 M 1902 74 IM
TANDOC JR, VALENTIN T, BETHEL CLINIC, 67114
283-3600
39 M 74809 62 U
TOMPKINS, CARL D, 316 OAK STREET, 67114
283-1380
22 M 1902 51 FP
VOGT, VERNON W, BETHEL CLINIC, 67114
283-3600
22 M 3005 53 FP
WEBER, ROY R, 215 S PINE, 67114
283-3025
46 M 1902 73 IM
WHEELER, DWIGHT E, BETHEL CLINIC, 67114
283-3600
50 M 2012 76 IM
WIENS, J WENDELL, 201 S PINE, 67114
283-3600
32 M 1902 59 GS

NORTON—913
(Northwest Kansas Society)

COLIP, F MERLYNN, 711 N NORTON, 67654
877-3305
35 M 1902 61 FP
COOPER, ARTHUR E, 305 W WILBERFORCE, 67654
-
08 M 1611 34 GP
HARTLEY, ROY WILEY, 711 N NORTON, 67654
877-3305
37 M 1902 63 FP
HARTMAN, ROGER L, 711 N NORTON, 67654
877-3305
35 M 1902 61 FP
LONG, ROBERT C, 711 N NORTON, 67654
927-3305
27 M 1902 53 GS
MEYER, JACK R, 711 N NORTON, 67654
877-3305
51 M 1902 76 FP

NORTONVILLE—913
(Shawnee County Society)

MADISON, WILLARD A, 66060
886-2110
20 M 1902 51 FP

OAKLEY—913 (Northwest Kansas Society)

OHMART, RICHARD V. PD BOX 756.67748
672-3262
36 M 1902 62 FP
SEKAVEC, GORDON B. 209 CENTER AVE.67748
672-3351
07 M 1902 38 FP

OBERLIN—913 (Northwest Kansas Society)

PALMER, MARGUERITE L. BOX 110.67749
475-2221
25 F 5404 54 GP
SIMPSON, ROBERT LIMBAUGH. 902 W COLUMBIA.67749
475-2221
25 M 4706 51 GS
WHITAKER, REN R. 902 W COLUMBIA.67749
475-2222
37 M 5404 66 FP

OLATHE—913 (Johnson County Society)

ARONOFF, MICHAEL E. 407 S CLAIRBORNE STE 209.66062
782-3953
39 M 1604 64 ENT
ASBURY, LAWRENCE J. OLATHE COMMUNITY HDSP.66062
782-1451
45 M 2878 77 EM
BEEBE, EDMER. 420 EAST CEDAR.66061
-
03 M 5605 32 FP
BLISS, JOY V. 42 HOLLY DRIVE.66062
782-2292
42 F 3005 68 ANES
BLUM, MICHAEL A. D.O.. 401 S CLAIRBORNE.66062
764-7060
47 M 2878 73 PD
BROWN, PAUL W. 407 S CLAIRBORNE.66062
782-7515
44 M 1902 70 FP
CHENOWETH, JOHN R. D.O.. 1003 STRATFORD RD.66062
-
36 M 2878 70 OST
CLEDENIN, ROBERT KEELE. 1950 E SANTA FE.66062
341-2100
48 M 1902 73 EM
CONANT, FERRILL R. 21325 W 180TH.66062
-
56 M 1902 86
DELPHIA, ROBERT E. 401 S CLAIRBORNE.66062
782-1610
24 M 1902 56 FP
EIDT, DAVID W. 407 S CLAIRBORNE.66062
782-8487
44 M 2501 70 FP
EIDT, LAURENCE A. 407 SOUTH CLAIRBORNE.65052
782-8487
44 M 1902 71 FP
FAILING, TRENT L. 527 PERSIMMON DR.66061
-
55 M 1902 87
FORTUNE, CEDRIC B. 405 S CLAIRBORNE.66062
782-3322
43 M 1902 66 FP
GLAZZARD, CHARLES D. 407 S CLAIRBORNE.66062
782-3384
28 M 2507 56 P
HALVORSON, HOWARD C. 407 S CLAIRBORNE.66062
782-2020
41 M 5404 66 U
HUDSON, ROBERT P. 12925 FRONTIER RD.66061
588-7040
26 M 1902 52 IM
JENSEN, THOMAS W. 407 S CLAIRBORNE.66052
782-1148
47 M 3005 73 ORS
LAIRD, DALE D. ONE PATRONS PLAZA.66061
782-3631
42 M 1902 68 OPH
MATHEWS, ROBERT C. 300 S ROGERS RD.66062
782-1451
50 M 2834 77 EM
MATTHEW, WILLIAM L. PD BOX 910.66061
782-3322
29 M 1902 56 FP

MCCANN, WILLIAM E. PD BOX 8.66061
782-0262
22 M 3901 48 FP
MEE, ADRIAN W. 28 HOLLY DRIVE.66062
782-2292
19 M 1902 54 ANES
MELENDRES, JUANITO M. 7400 GLEASON RD - RR 1.66061
684-4649
36 M 74809 61 PD
MENDLICK, R MICHAEL. 407 S CLAIRBORNE STE 101.66062
782-1148
44 M 1902 70 ORS
MORGAN II, DAVID LLOYD. 807 S CLAIRBORNE.66062
782-8300
49 M 2820 77 IM
NOTTINGHAM, ROBERT M. 401 CLAIRBORNE.66062
782-1610
49 M 1902 78 FP
OYLER, JONATHAN M. 1706 PENROSE.66062
-
59 M 1902 87
PIERRON, GEORGE J. 540 EAST SANTA FE.66061
782-0260
22 M 1902 47 FP
RONOVDO, STEVEN A. 300 SOUTH ROGERS RD.66062
782-2292
47 M 1902 73 ANES
RUHLIN, JAMES L. 807 S CLAIRBORNE.66062
782-8300
46 M 1902 72 IM
SEAMAN, LAUREN I. 1613 E SHERIDAN.66062
-
07 M 6001 38 FP
SETTLE JR, RUSSELL D. 407 S CLAIRBORNE.66062
782-3384
35 M 1902 60 P
SHANKER, STUART G. 401 S CLAIRBORNE STE 200.66062
764-7060
49 M 2803 75 PD
SHEFFER, KEITH D. 407 S CLAIRBORNE STE 101.66062
782-1148
37 M 1720 67 ORS
SNYDER JR, RICHARD HENRY. 300 S ROGERS ROAD.66061
782-1451
45 M 1902 73 ANES
STITES III, HAROLD W. 807 CLAIRBORNE.66062
782-8300
50 M 2803 80 IM
THOMPSON, SCOTT W. 405 CLAIRBORNE. 66061
782-3322
51 M 64914 78 FP
YEDMANS, RONALD N. 405 S CLAIRBORNE SUITE 4.66062
782-3073
40 M 1902 67 DBG

ONAGA—913 (Pottawatomie County Society)

FLECKENSTEIN, CHARLES S. 501 LUCIEN ST.66521
-
07 M 1902 36 OO
WALSH, THOMAS E. ONAGA CLINIC.66521
889-4241
48 M 1902 74 FP

OSAGE CITY—913 (Flint Hills Society)

ADAMS, DWIGHT. 608 HOLLIDAY.66523
528-3161
M 1902 3P
WILLIAMS, HOMER J. 611 S SIXTH.66523
-
05 M 1902 31 FP

OSAWATOMIE—913 (Miami County Society)

APPENFELLER, WILLIAM D. 524 BROWN AVE.66064
755-3166
25 M 1902 53 FP

OSWEGO—316
(Labette County Society)

BURGESS, ARTHUR P., 504 5TH STREET, 67356
795-4427
19 M 1902 52 FP

OTTAWA—913
(Franklin County Society)

BLANKENSHIP, JIM D., 1320 S ASH, 66067
242-5581
50 M 2834 76 FP
CORDER, S SCOTT, 1502 S CEDAR, 66067
242-2641
51 M 1902 76 FP
GOLLIER, ROBERT A., 66067
-
13 M 1902 37 00
GOLLIER II, ROBERT A., 1320 S ASH, 66067
242-1620
40 M 1902 66 FP
HADLEY, DELMONT C., 1320 SOUTH ASH, 66067
242-3891
35 M 1902 64 FP
HENNING, CALVIN W., 1502 CEDAR, 66067
-
05 M 1902 35 FP
LAURY, DAVID G., 1320 S ASH, 66067
242-1620
17 M 1606 44 FP
PHILGREEN, DONALD E., 1320 S ASH, 66067
242-3891
39 M 1602 67 FP
RANDM, WILLARD B., 1320 S ASH, 66067
242-1620
49 M 1902 77 FP
REYES JR, FRANCISCO A., 1320 S ASH, 66067
242-5312
38 M 74801 61 GS
SPEER, LOUIS N., PO BOX 0, 66067
242-1257
14 M 1606 41 FP
STREHLow, CHESTER H., PROFESSIONAL PLAZA BLDG, 66067
242-3891
30 M 1902 57 FP

OVERBROOK—913
(Shawnee County Society)

RUBLE JR, JAMES L., OVERBROOK COMM CLINIC, 66524
665-2205
26 M 1902 53 FP

PAOLA—913
(Miami County Society)

BANKS, ROBERT E., PO BOX 298, 66071
294-2305
29 M 1902 55 FP
ROWLETT, JACK G., PO DRAWER A, 66071
294-2356
21 M 1902 52 FP
STANLEY, REX C., PO DRAWER A, 66071
294-2056
24 M 1902 52 GS

PARSONS—316
(Labette County Society)

AVES, AGNES, 1509 MAIN, 67357
421-0600
38 F 74801 59 IM
AVES, RENATO B., 1509 MAIN STREET, 67357
421-0600
35 M 74801 59 GS
CAREY, LARRY J., LABETTE CO MED CL SUITE 5, 67357
421-8361
51 M 1902 74 FP

CRAMER, GUY W., 412 MURDOCK, 67357

-
11 M 1902 39 00
DAIZ, ANTONIO S., 1721 CORNING, 67357
421-0091
37 M 74810 63 OR
DILLON, WILLIAM L., LABETTE CO MED CL BOX H, 67357
421-0881
45 M 1902 71 ORS
HENDERSON, CHARLES F., 1617 GRAND, 67357

-
14 M 1902 40 00
KISHORE, ROY N., LABETTE CLINIC #3, 67357
421-4062
44 M 49511 66 OTO
KISHORE, SHEELA, 3604 GABRIEL, #222, 67357
421-2741
43 F 49511 66 ANES
LAVA, CHIRUND, PO BOX 290, 67357
421-6210
40 M 89102 63 GS
MARTIN, EARL A., 1516 GRAND, 67357

-
07 M 1606 35 00
MILLER, CHARLES H., 2819 CLARK, 67357

-
07 M 3006 32 00
MILLER, DEAN M., 203 CRESTVIEW, 67357
421-4880
22 M 1902 48 R
MILLER, STEPHEN FRANCIS, 1509 MAIN, 67357
421-0600
45 M 1902 70 GS
PACE, JOHN D., KATY HOSPITAL CLINIC, 67357

-
94 M 1902 20 FP
PAI, RADHA V., PO BOX 1057, 67357
421-0080
45 F 6701 ANES
PAI, VARADARAJ S., PO BOX 1057, 67357
421-0080
42 M 6701 U
PARANJITHI, SUBRAMONIAM P., 1509 MAIN, 67357
421-6160

39 M 49531 64 IM
PAULS, DANIEL N., PO BOX 1014, 67357
421-1431
45 M 1902 71 IM
ROTHSTEIN, TERRY B., 220 NORTH 32ND, 67357
421-5900
43 M 1606 69 OPH
SHARMA, ARUN L., 1509 MAIN, 67357
421-0600

46 F 49503 69 FP
TANG, CHANTRA, PO BOX 1054, 67357
421-2460
47 F 89104 71 PD
TANG, SAROHD, PO BOX 1054, 67357
421-2460
43 M 89102 69 OBG
VERMA, ASHA, 400 KATY, 67357
421-9090

37 F 49530 63 PD
VICHYANOND, PAKIT, LABETTE CO MEDICAL CENTER, 67357
421-2460
59 M 89101 75 PD
WHITE, JOHN P., PARSONS CLINIC, 67357
421-0600
17 M 1902 42 FP

PEABODY — 316
(Harvey County Society)

CRIDER, K L., 500 W FOURTH, 66866
983-2131
F FP

PITTSBURG—316
(Crawford County Society)

ARMSTRONG, HAROLD J., PROFESSIONAL BUILDING, 66762
232-2600
40 M 1902 68 ORS
BENA, JAMES H., 109 EAST 9TH, 66762
231-6950
12 M 3005 36 PD
BERKEY, VERNON A., KIRKWOOD BLDG, 66762
231-7650
18 M 1902 43 R

BIERLEIN, KENNETH J. 812 S CATALPA.66762

36 M 1606 33 00
COOMER, TYLER E. 315 NATL BANK BLDG.66762

231-7730
30 M 2101 59 GS
COOPER, KENT J. 909 CENTENNIAL.66762

231-6280
41 M 1902 73 FP
ERICKSON, CLARENCE W. 217 NATL BANK BUILDING.66762

231-7400
06 M 1902 33 1M
ESCH, JOHN G. 207 KIRKWOOD BLDG.66762

231-5360
24 M 3006 48 GS
FREEMAN, MALCOLM C. 1102 VILLAGE DR.66762

231-6100
44 M 35207 68 ANES
GOWETZ, MOESTO S. 909 E CENTENNIAL #6.66762

231-2490
35 M 72601 63 PD
HOLSINGER, DONALD M. 1015 MT CARMEL PL.66762

231-5900
38 M 1902 64 1M
HUEBNER, ROBERT STEPHAN. NATIONAL BANK BLDG.66762

231-6160
42 M 1606 67 GS
HUERTER, DAVID F. 909 CENTENNIAL.66762

231-1650
46 M 1902 72 1M
LANCE, RAYMOND W. 608 W QUINCY.66762

22 M 1902 47 00
LEFFLER, PAUL B. 309 WINWOOD.66762

02 M 1902 40 00
MILLER, EARL E. 1312 S BROADWAY.66762

231-6410
13 M 1902 37 ENT
MULLER, SAMUEL B. 611 W QUINCY.66762

05 M 1902 34 00
NEWMAN, CLIFFORD B. 1204 E 7TH.66762

01 M 1902 28 00
ODGERS, ROONEY K. 909 CENTENNIAL.66762

231-4300
M 1902 74 1M
PAPP JR, S DEAN, R 5 BOX 293.66762

231-7650
46 M 1902 72 DR
PARSI, MANUTCHEHR K. 909 CENTENNIAL.66762

231-3770
38 M 51701 64 DBG
POGSON, GEORGE W. 1015 MT CARMEL PLACE.66762

231-5900
24 M 1902 47 1M
RAMIREZ, AUGUSTO H. 909 CENTENNIAL.66762

231-1600
32 M 26407 58 1M
RAMIREZ, IRENE. 909 CENTENNIAL.66762

231-6280
F PD
SCHLEMMER, ROGER B. 1009 S BROADWAY.66762

231-6380
37 M 1902 68 JPH
SEGLIE, FLOYD ROYALD. 909 CENTENNIAL DR SUITE 3.66762

231-6280
43 M 1902 69 FP
TWEET, FREDRICK A. RR 5 BOX 196.66762

231-6100
39 M 1602 66 PATH
WOOD, DOUGLAS H. 413 W JEFFERSON.66762

11 M 5605 36 00
YAGHMOUR, TALAAT E. 2701 S ROUSE.66762

231-0850
40 M 33002 64 U
ZABEL, KENNETH P. 909 CENTENNIAL.66762

231-1650
37 M 1902 65 1M

PLAINVILLE—913
(Central Kansas Society)

KELLERMAN, RICK. 409 S COCHRAN.67663
434-4609

M 1902 FP
PAGE, D VALE. 409 S COCHRAN ST.67663
434-4609

20 M 1902 51 FP

PEDERSON, ARNOLD M. 409 S COCHRAN.67663

434-4609
22 M 1902 51 FP

PLEASANTON—913
(Bourbon County Society)

JUSTUS, WILLIAM J. .66075

352-6134
29 M 1902 55 FP

PRATT—316
(Ninnescah Society)

AMBLER, CARL O. 200 COMMODORE.67124

672-6476
31 M 1902 57 R

BARKER, PATRICK N. 420 COUNTRY CLUB RD.67124
672-7411
45 M 1902 71 GS

BLACK, CYRIL V. 223 E 4TH.67124
672-6403

05 M 4802 30 GS
OILLON, STEVEN C. 420 COUNTRY CLUB RD.67124

672-7411
53 M 1902 78 1M

FILLEY, VERNON W. 310 E 2ND.67124
572-5555

13 M 3005 43 GS
FREEMAN, F GILES. 310 E 2ND.67124

672-5555
18 M 1902 44 FP

GARD, RAYMOND F. 593 TERRACE DR.67124
-

01 M 1902 27 GS
PITMAN, WILL D. 717 WEST 3RD.67124

-
98 M 1902 25 00

QUENZER, RONALD W. 420 COUNTRY CLUB RD.67124
672-7411

46 M 1601 73 1M

SIBALA, JUSTO L. 200 COMMODORE.67124
672-7159

20 M 74802 49 R
SUITER, DANIEL JAY. 420 COUNTRY CLUB.67124

672-7411
44 M 1902 71 GE

THORPE, FRANCIS A. 2 LAKE ROAD.67124
672-5555

08 M 1606 35 FP
WARD, ROBERT L. 420 COUNTRY CLUB.67124

672-7411
24 M 1902 52 FP

WOLFF, FREDERICK P. 223 E 4TH.67124
672-6403

20 M 1902 44 1M

PROTECTION—316
(Iroquois County Society)

GLENN, LYLE G. 146 BROADWAY BOX 447.67127

622-4586
12 M 1606 40 FP

QUINTER—913
(Northwest Kansas Society)

GUNTER, CARL C. QUINTER CLINIC BLDG.67752

754-3333
20 M 1902 51 FP

HIESTERMAN, HERMAN W. QUINTER CLINIC BLDG.67752
754-3333

23 M 1902 51 FP

RANSOM—913
(Central Kansas Society)

MCLAIN, KENNETH. BOX 247.67572

731-2295
21 M 1902 46 FP

RUSSELL—913
(Central Kansas Society)

MERKEL, EARL O. SHIELDS BLDG. 67665
483-2178
32 M 1902 57 FP
PANICHABHONGSE, SAMBUNDH, 213 WEST 7TH, 67665
493-2133
43 M 89101 67 GS
PETTIGORN, WALTER J. 624 W 12TH, 67665
-
12 M 1902 37 OO
STARKEY, JERALD L. 326 MAIN, 67665
483-2178
30 M 1902 56 FP
SWANN, CLAIR L. 112 W 51ST, 67665
493-4212
13 M 1902 39 1M
WHITE, FAGAN N. 356 W 5TH, 67665
-
11 M 702 36 OO

SABETHA—913
(Northeast Kansas Society)

KENNALLY, KEVIN P. 1115 MAIN, 66534
284-2141
53 M 1902 78 FP
MONTGOMERY, THOMAS ALLEN, 1013 WYOMING, 66534
-
10 M 1902 49 OO
WENGER, GREGG O. 1115 MAIN, 66534
284-2141
M 1902 78 PO
YULICH, JOHN O. PO BOX 227, 66534
284-2125
33 M 1902 59 FP

SALINA—913
(Saline County Society)

ALLEN, MONTE L. 600 S SANTA FE, 67401
927-0307
36 M 1902 61 OTO
ALSOP, WILLIAM R. 737 E CRAWFORD, 67402
827-7261
52 M GE
ANDERSON, JOOY, 737 E CRAWFORD, 67402
827-7261
32 F 1902 59 1M
BAXTER, W REESE, P D BOX 1707, 67401
825-8221
47 M 1902 73 FP
BELL, MARK G. 909 E WAYNE, 67401
223-7225
50 M ENT
BROWN, ROBERT WAYNE, PO BOX 1747, 67402
825-7251
23 M 1902 55 1M
BRUMMETT, RICHARD R. P O BOX 1707, 67401
825-8221
34 M 1902 64 FP
CATHCART-RAKE, WILLIAM F. 737 E CRAWFORD, 67402
827-7261
48 M 1902 74 1M
CLARK, DAVID H. 617 E ELM, 67401
825-8221
36 M 1902 62 FP
COFFEY, RYD B. 671 ELMORE DRIVE, 67401
823-6397
24 M 1902 47 ORS
CONNELLY, MAURICE R. RR 3 BOX 44, 67401
-
12 M 2002 38 OO
CONNER, BRIAN, 1518 B EAST IRON, 67401
825-2272
46 M 1902 JPH
COSSETTE, JERROLD E. 909 E WAYNE, 67401
823-7225
46 M 1902 75 ENT
COVERT, THOMAS J. 737 E CRAWFORD, 67402
827-7261
M PO
CULTRON, FRANK T. 800 E CRAWFORD, 67401
823-8151
10 M 1643 38 OPH

D'SOUZA, BISMARCK C. BOX 2318, 67401
827-9526
45 M 49501 67 R
DOWELL, JAMES C. 645 E IRON, 67402
827-7255
26 M 1611 49 1M
DRAENEL, H RICHARD, 600 S SANTA FE ST, 67401
827-0307
18 M 1902 53 OTO
DREHER, HEVRY S. 737 E CRAWFORD, 67402
827-7261
15 M 1902 43 1M
EATON, GLEN E. RR 6 BOX 359, 67401
827-3064
28 M 1902 54 ANES
EATON, LESLIE F. RR 1 BOX 346, 67401
-
06 M 1902 32 OO
ELLISON, PAUL O. 1499 E IRON, 67401
825-7271
35 M 2105 60 OPH
FEIGHNY, ROBERT E. 2437 VILLAGE, 67401
-
20 M 1902 51 ORS
FORSTER JR. LOUIS G. BOX 1747 - 130 W CLAFLIN, 67401
825-8221
47 M 1902 73 FP
FRANCIS, ANTHONY E. 519 S SANTA FE, 67401
827-4424
54 M 1902 77 ORS
FREEMAN, RAYMOND S. 737 E CRAWFORD, 67402
827-7261
20 M 702 50 PD
GANS, FREDERICK A. 950 S ELEVENTH, 67401
-
22 M 2834 46 OD
GRIFFITH, FRANK H. 1493 E IRON, 67401
827-0488
45 M 4813 75 OPH
GUNN, MARVIN R. BOX 2318, 67401
827-9526
28 M 3901 54 R
GUZMAN, MANUEL, CKMHC, 67401
823-6322
27 M 64901 54 P
HARBIN, GARY LYNN, 523 S SANTA FE, 67401
823-7213
50 M 1902 75 ORS
HARRIS, NORMAN R. 430 S 7TH, 67401
825-8191
30 M 1902 59 OBG
HASSLER, RANDY D. 645 E IRON, 67401
827-9635
45 M 1902 71 U
HATTON, LLOYD W. 709 HIGHLAND, 67401
-
06 M 1902 33 P
HERRMAN, ADAM L. 519 S SANTA FE, 67401
827-4424
48 M 1902 74 ORS
HESSE, FREDERICK J. 135 E CLAFLIN, 67401
827-9631
50 M 1902 75 1M
HODGES, MERLE A. 430 S 7TH ST, 67401
825-8191
34 M 1902 58 OBG
HOLMAN, JON B. PO BOX 61, 67401
827-9366
33 M 1902 63 P
HUNNINGHAKE, RONALD, 617 E ELM BOX 1707, 67401
825-8221
51 M 1902 76 FP
JACKSON JR. OELMAS A. 645 E IRON, 67402
827-7255
35 M 2101 60 1M
KREHRIEL, MARK A. 617 E ELM, 67401
825-8221
49 M 1902 74 FP
KRUCKEMYER, ALAN L. 645 E IRON, 67401
823-2215
45 M 1103 71 ORS
LACE, MAX S. 124 S OAKDALE, 67401
-
19 M 3005 43 OO
LASLEY, DAVID A. 645 E IRON, 67401
-
22 M 1606 47 JJ
LAWRENCE, GILBERT A. 116-A S SEVENTH, 67401
827-9526
M R
LIVINGSTON, CHARLES E. 400 E IRON, 67401
823-9166
32 M 1611 57 GS

LUNGSTRUM, JACK E, PO BOX 1346,67401
823-2215
21 M 1902 59 DRS
MACY, NORMAN E, BOX 1285,67401
827-4053
35 M 1902 60 PATH
MACY, TEO L, PO BOX 360,67402
827-7261
43 M 1902 71 GS
MARCHBANKS, DONALD L, 520 COUNTRY CLUB RD,67401
923-2380
24 M 1902 51 FP
MARSHALL, GEORGE W, PO BOX 1705,67401
225-3191
44 M 1902 70 DBG
MARTIN, OLIVER L, 715 E REPUBLIC,67401
827-9631
08 M 1902 37 DBG
MATHIS, JERRY L, 2000 GLENDALE,67401
827-9343
35 M 1902 62 PDA
MAXWELL, GORDON E, 135 E CLAFIN,67401
827-9631
29 M 1902 55 DBG
MCCRAE, SPENCER C, 519 S SANTA FE,67401
-
18 M 3509 43 DD
MILLER, ELOEN V, 1928 RIDGELEA,67401
827-3061
19 M 1902 44 ANES
MITCHELL, JOHN C, 116 W LAKE DR,67401
827-3061
13 M 1902 38 GS
MOWERY, WILLIAM E, PO BOX 360,67402
827-7261
23 M 1902 47 GS
NEUMANN, JAMES W, 600 S SANTA F,67401
825-5041
M N
NICKELL, WENDELL K, 400 E IRON,67401
823-9166
26 M 1606 50 TS
NIXON, RICHARD R, BOX 2318,67401
927-9526
32 M 1643 57 R
NULL, WILLIAM G, 135 E CLAFIN,67401
827-9631
31 M 102 57 PD
OBAYOOD, GUILLERMO, 2110 KNOXCREST DR,67402
827-7261
35 M 26404 62 R
PALMER, GERALD K, PO BOX 1285,67401
923-7201
24 M 1803 53 PATH
PARKS, DOUGLAS S, 2158 KENSINGTON,67401
-
56 M 1902 83 FP
PETERSON, JAMES E, BOX 2318,67401
827-9526
53 M 1902 78 DR
REECE, RICHARD J, BOX 2318,67401
827-9526
23 M 1902 49 R
RODERICK, JAMES E, 645 E IRON,67401
827-9635
23 M 1902 47 U
ROMEISER, REX S, 645 E IRON,67401
827-9635
41 M 1902 67 U
RUEB, ANDREW E, 11 CRESTVIEW DR,67401
827-6691
11 M 1606 35 GS
SCANLAN, TIMOTHY M, PO BOX 1747,67402
825-7251
46 M 2604 71 FP
SCHMIDT, RAMON WARNER, 400 E IRON,67401
823-9166
39 M 1902 65 GS
SCOTT, CHESTER E, 519 S SANTA FE,67401
827-5549
23 M 1902 51 FP
SEATON, ROBERT D, PO BOX 360,67402
827-7261
M NEP
SEBREE, STEVEN G, PO BOX 360,67401
827-7261
47 M 1902 73 DBG
SLOO, MILD G, 645 E IRON,67401
823-2215
41 M 1902 67 DRS
SMITH, BOYD E, BOX 1285,67401
827-4053
46 M 3005 72 PATH

SMITH, DAVID E, PO BOX 360,67402
827-7261
50 M 1902 76 GS
SMITH, HAROLD R, PO BOX 360,67402
827-7261
19 M 1902 51 GS
SNYDER, THOMAS E, 737 E CRAWFORD,67402
827-7261
47 M 1902 DBG
STOSKOPF, LAWRENCE E, 2413 EDGEHILL,67401
323-9498
39 M 1902 72 ANES
STUEWE, BRADLEY R, 737 E CRAWFORD,67402
827-7261
49 M 1M
TAYLOR, THOMAS F, 430 S OHIO,67401
827-0346
26 M 1902 53 FP
WAGENBLAST, HOWARD R, 737 E CRAWFORD,67402
827-7261
21 M 1902 49 FP
WATERS, CLARENCE N, 530 S 5TH,67401
823-6497
13 M 2834 48 0
WEBER, ROBERT W, 645 E IRON,67402
827-7255
26 M 1902 49 1M

SATANTA—316
(Southwest Kansas Society)

JABEL, JUVENAL T, SATANTA CLINIC,67870
649-2771
43 M 74809 1M
TADURAN, VIRGILIO, SATANTA CLINIC,67870
649-2771
43 M 74810 43 PATH

SCOTT CITY—316
(Southwest Kansas Society)

DUNN, DANIEL R, 202 COLLEGE,67871
872-2187
49 M 1902 74 FP
HOPKINS JR, B MORRISON, 202 COLLEGE,67871
872-2187
23 M 1902 53 FP

SEDAN—316
(Southeast Kansas Society)

TAYLOR, ELMER W, 120 WEST OSAGE,67361
725-3141
28 M 512 57 FP
WALKER, WILLIAM K, 111 E CHEROKEE,67361
725-3171
18 M 1902 45 FP

SENECA—913
(Northeast Kansas Society)

BERKLEY, NORMAN W, 15 SOUTH 5TH ST,66538
336-2128
31 M 1902 63 FP
GILBERT, J HOWARD, 211 S FOURTH,66538
-
05 M 1902 41 DD

SHARON SPRINGS—913
(Northwest Kansas Society)

CHUNG, JOHN J, WALLACE CO MEO CLINIC,67758
852-4214
23 M 58301 48 FP

SHAWNEE MISSION—913
(Johnson County Society)

AMUJA, DEEPAK, 12310 OVERBROOK CT,66209
-
62 M 1902

ALLEN, MAX S. 5103 W 96TH TERR.66207
 588-5063
 11 M 1902 37 IM
 ALTENBERND, ELVIN CONRAD, 7319 W 81ST.66204
 648-2010
 26 M 1902 54 FP
 ALVAREZ, LUIS A. 10211 MISSION RD.66208
 642-3400
 32 M 64914 61 FP
 ARMBRUSTER, ALBERT A. 9119 WEST 74TH SUITE 202.66204
 352-9220
 17 M 512 55 GS
 ARONSON, RITA K. 4892 SKYLINE DR.66205
 -
 56 F 1902 87
 ATHON, MERRILL D. 6806 W 83RD.66204
 642-4242
 24 M 1902 54 FP
 ATKINS, JEFFREY A. 6648 CHARLES.66216
 -
 61 M 1902 87
 BADEEN II, LOUIS JOHN. 10550 QUIVIRA RD.66215
 492-3344
 49 M 2846 74 DPH
 BAEKE, JOHN D. 6806 WEST 83RD.66204
 642-4242
 19 M 1902 52 FP
 BAKER, WILLIAM STEVEN, 7700 W 63RD.66202
 262-1843
 47 M 702 73 D
 BALANOFF, ARNOLD Z. 4601 W 109TH SUITE 122.66211
 642-4040
 42 M 1803 67 PD
 BANSAL, ROOPA D. 8901 W 74TH SUITE 147.66204
 384-2220
 37 F 49504 64 FP
 BANSAL, SATISH C. 8901 W 74TH SUITE 147.66204
 384-2220
 38 M 49541 61 ORS
 BAPTIST, JEREMY E. 5811 OUTLOOK.66202
 432-0625
 40 M 2846 78 A
 BARE II, CHARLES E. 8901 W 74TH SUITE 353.66204
 677-2460
 43 M 1902 69 U
 BARKER, ELIZABETH B. 4121 WEST 83RD SUITE 123.66208
 381-6669
 30 F 4706 55 P
 BARKER, JAMES BERTON, 8901 W 74TH.66204
 362-6310
 31 M 4706 55 OTO
 BARNETT JR, THOMAS E. 10550 QUIVIRA SUITE 290.66215
 492-2555
 52 M 1902 75 IM
 BARNHART, RONALD J. 9119 WEST 74TH.66204
 831-2334
 41 M 2501 68 OBG
 BARNHORST, DONALD A. 10550 QUIVIRA STE 510.66215
 492-6200
 37 M 2834 63 CDS
 BARR, RICHARD N. 7301 MISSION ROAD.66208
 432-4366
 32 M 1902 57 OPH
 BARRICK, BRUCE, SH MISSION MEDICAL CENTER.66201
 676-2340
 39 M 1902 65 PATH
 BATTY, LARRY H. 9119 W 74TH.66204
 831-2334
 51 M 1902 77 OBG
 BATTY, THOMAS V. 5555 W 58TH.66202
 432-2080
 21 M 3806 54 FP
 BAUER, JOSEPH G. 9640 CATALINA.66207
 -
 59 M 1902 88
 BAUER, LAKE W. 4818 W 80TH.66208
 542-5369
 20 M 1902 49 ANES
 BAUER, MARTIN L. 2500 W 71ST.66205
 281-8881
 47 M 1902 73 PD
 BECK JR, CALVIN E. 6130 MISSION.66208
 -
 59 M 1902 87
 BEILMAN, GREG J. 10577 RILEY.66212
 -
 59 M 1902 86
 BELL, DELORIS W. 4601 W 109TH SUITE 116.66211
 341-6550
 42 F 1902 68 OPH
 BELT, ROBERT J. 8901 W 74TH SUITE 34.66204
 362-3023
 45 M 702 71 IM
 BELZER, EDWARD G. PO BOX 7426.66207
 381-8282
 36 M 3005 58 PD
 BERRY, JOHN M. 8800 W 75TH SUITE 320.66204
 262-3288
 47 M 1902 74 D
 BIKALES, VICTOR WILLIAM, 10688 RIGGS LANE.66212
 384-1311
 13 M 2105 38 P
 BILLINGSLEY, THAD M. 7000 SQUIBB RD #100.66202
 432-9900
 41 M 1902 66 D
 BISHOP, FRANCIS E. 7501 MISSION RD.66208
 648-3533
 20 M 1902 45 P
 BISHOP, HENRY R. 10550 QUIVIRA SUITE 320.66215
 492-3600
 53 M 4813 79 OBG
 BLETZ, DONALD B. 10550 QUIVIRA SUITE 510.66215
 492-6200
 28 M 5104 58 IM
 BLES, J MICHAEL, 5949 NIEMAN ROAD.66203
 631-1300
 35 M 1902 61 FP
 BRAVERMAN, DAVID ELLIOTT, 4601 W 109.66211
 341-1101
 47 M 2507 72 PDD
 BROOKS, DOUGLAS, 6313 W 50TH.66202
 -
 51 M 1902 87
 BROUCEK, FRANCIS J. 4121 W 83RD.66208
 642-6845
 32 M 1643 58 P
 BROWN, WILLIAM R. 7301 MISSION RD.66208
 236-8866
 23 M 1902 48 IM
 BROXTERMAN, STEVEN JOSEPH, 8901 W 74TH SUITE 225.66204
 362-5510
 51 M 1902 76 FP
 BRUNER JR, KENNETH W. 4505 W 64TH.66208
 262-3286
 44 M 2401 70 PATH
 BRUNGARDT, BERNARD A. 4505 W 64TH.66208
 -
 21 M 3006 46 OD
 BRUNING, ROGER MARION, 7301 MISSION SUITE 342.66208
 384-0745
 48 M 1902 70 FP
 BUCK JR, WILLIAM D. 10807 JASIS CT APT 113.66203
 -
 59 M 1902 86
 BUCKMAN, MARTIN SPALDING, 10550 QUIVIRA SUITE 290.66215
 492-2555
 49 M 2802 76 IM
 BUHR, BRUCE R. 9800 W 56TH.66203
 -
 51 M 1902 87
 BURGER, PAUL B. 5638 NIEMAN RD.66203
 631-6114
 25 M 2834 50 FP
 CALKINS, LARRY L. 5635 SUWANEE RD.66205
 -
 18 M 1902 43 DD
 CAMARATA, PAUL J. 12000 W 66TH.66216
 -
 60 M 1902 86
 CARDUFF, JAY J. 6300 GLENWOOD.66202
 432-4480
 25 M 3006 54 FP
 CARRILLO, BELINDA A. 9417 NIEMAN RD.66214
 -
 54 F 1902 87
 CASTEEL, CHARLES K. 8901 W 74TH SUITE 357.66204
 831-1003
 34 M 3901 59 U
 CATTANED, ERNEST A. 6100 MARTWAY.66202
 262-3930
 39 M 1902 65 IM
 CAVITT, ROBERT F. 9119 WEST 74TH.66204
 831-0700
 24 M 1902 48 GS
 CEDERLIND, CRANSTON JAY, 8901 W 74 SUITE 36.66204
 236-6455
 45 M 1902 71 OBG
 CHALABI, PHILLIP M. 11814 W 66TH.66203
 -
 61 M 1902 87
 CHANG, SHU FANG, 10200 W 75TH.66204
 432-7885
 25 F 24239 49 P
 CHRISTIAN, STANLEY J. 9129 DELMAR.66207
 -
 19 M 1902 44 OD

CLOYD, DAVIO W. 8901 W 97TH TERR.66212

55 M 3005 80 GS
CLYMER, DAVIO J. 2309 W 71ST TERR.66208

53 M 4705 80 ORS
COE, RICHARD D. 7301 MISSION RD SUITE 247.66208

362-8505
31 M 4804 56 OPH
COHEN, MARC D. 8800 W 75TH SUITE 300.66204

362-1226
51 M 1001 77 IM
COHEN, ROBERT A. 3700 W 83RD.66208

642-2100
39 M 2803 64 PD
COHN, STEVEN G. 8627 LINDEN DR.66207

334-2500
41 M 1902 67 ANES
COLEMAN, ROBERT L. 8901 W 74TH SUITE 1.66204

352-0100
41 M 4113 66 PS
CODLEY, DAVIO A. 8800 W 75TH SUITE 300.66204

362-1226
40 M 2802 66 RHU
COOPER, JACK R. 7301 MISSION RD SUITE 330.66208

432-6300
17 M 3840 43 VS
CORBIN, MURRAY D. 10550 QUIVIRA RD-5TH FL.66215

492-6200
39 M 1902 65 CO
COULTER, HENRY F. 4203 W 151 ST.66224

23 M 1902 51 DO
COULTER, THOMAS B. 8800 W 75TH SUITE 310.66204

677-3113
38 M 1205 64 OPH
COX JR, IRA. 5829 WOODSON RD BOX 975.66201

722-1100
19 M 1902 49 FP
CREEK, ALAN D. D.O.. 10550 QUIVIRA SUITE 430.66215

888-0777
46 M 2878 78 FP
CROW, JIMMIE R. 7312 ANTIOCH.66204

384-2850
53 M 1902 78 GS
CURRAN, KEVIN E. 4121 W 83RD.66208

649-9383
39 M 2803 65 OPH
CURTIS, JEFFERY L. 7711 HARDY.66204

55 M 1902 81 IM
DAVIS, RICHARD E. 8500 W 110TH SUITE 308.66210

648-5303
26 M 1902 54 P
DEAY, CHARLES T. 7505 FLINT.66214

60 M 1902 86
DELP, MAHLON M. 6131 TERRYDALE RD.66202

03 M 1902 34 IM
DEMISON, TERRY R. 5811 OUTLOOK.66202

432-0625
29 M 1902 56 PD
DERRINGTON, KENNETH L. FOX HILL MED BLDG.66211

341-3535
44 M 1902 71 FP
DOCKHORN, ROBERT J. 5300 W 94TH TERR.66207

381-4674
34 M 1902 60 POA
DOHERTY, WILLIAM R. 7600 STATE LINE.66208

649-3900
20 M 3006 56 FP
DUCKETT II, THOMAS G. 4601 W 109TH SUITE 116.66211

648-1022
41 M 1902 67 OPH
EIKERMANN, WILLIAM C. 9400 MISSION RD.66206

642-5184
42 M 1902 69 D
ENDERS, WRAY. 9034 COTTONWOOD DR.66215

02 M 1902 36 ANES
ESRIG, HAROLD L. D.O.. 8132 SAGAMORE.66206

381-5033
30 M 2878 60 ANES
ETZENHOUSER III, RUSSELL D. P O BOX 7426.66207

381-8282
34 M 1902 59 PD
EVANS, CAROL ANN. 6100 MARTWAY SUITE 2.66202

362-0000
54 F 2846 78 IM
EVANS JR, WILLIAM E. 8741 HIGH DRIVE.66206

362-7363
24 M 1902 58 FP

FERGUSON, ROBERT LEON. 8901 W 74TH SUITE 208.66204

362-0300
47 M 1902 73 IM
FORDYCE, NORMAN. 8901 W 74TH.66204

722-0020
41 M 1902 67 OTJ
FRANCISCO, CARENCE L. 3509 W 85TH.66206

371-6802
09 M 1902 34 ORS
FULLEN, JERYL G. 8901 W 74TH SUITE 124.66204

831-2604
43 M 401 68 ORS
GALBUT, ALAN S. 6720 W 52ND PLACE APT 38.66202

55 M 3806 81 IM
GARCIA, FRANCISCO. 8020 SANTA FE.66204

642-5000
32 M 27501 60 FP
GARCIA-FERRER, CIRA M. 9409 W 82ND.66204

60 F 1902 87
GARDINER, ROBERT C. 7601 GARNETT APT 10.66214

57 M 1902 86
GARDNER, GLENN M. 5200 WEST 64TH.66208

35 M 2803 60 IM
GAUGHAN, MICHAEL J. 7312 ANTIOCH SUITE 150.66204

677-0883
49 M 1902 74 R
GENTRY, KALE C. 5105 W 84TH.66207

632-4242
31 M 1902 60 FP
GERRON, LINDA L. 5201 HOWE DR.66205

53 F 1902 86
GILBERT, ROBERTA M. GEORGETOWN MEDICAL BLDG.66204

362-4040
35 F 3506 62 D
GILLEN, BILLY A. 8802 BIRCH LANE.66207

29 M 1902 54 ANES
GOLSTEIN, GERALD L. 4601 W 109TH SUITE 318.66211

383-3630
47 M 16504 76 D
GOLLERKERI, MOHAN P. 7301 MISSION RD STE 339.66208

236-8866
30 M 49516 52 HEM
GOMEZ, FRANCISCO. 4200 SOMERSET #160.66208

649-7300
15 M 26401 43 P
GOOD, WENDELL Lisle. 4601 W 109TH.66211

649-3883
24 M 1902 48 FP
GOSALIA, ANIL V. 11701 MACKEY.66210

334-2500
46 M 49501 64 GP
GRAHAM, SUSAN B. 6423 RILEY.66202

03 M 1902 34 IM
GRASHOFF, JOYCE A. 9138 W 102ND TERR.66212

596-4180
59 F 3005 80 EM
GREENE, LAWRENCE S. 10550 QUIVIRA RD STE 510.66215

492-6200
33 M 3506 54 GE
GRIESHABER, GORDON J. 5206 SYCAMORE.66205

54 M 1902 86
GROSSMAN, HARVEY M. 4601 W 109TH SUITE 122.66211

642-4040
49 M 1902 74 PD
GRUNOMEIER, ANNETTE M. 10550 QUIVIRA SUITE 250.66215

492-2013
46 F 1611 77 PD
HACKER, DAVID CHARLES. 6900 W 67TH.66202

588-6670
50 M 1902 75 ANES
HAMTIL, LAWRENCE W. 10550 QUIVIRA RD.66215

341-3937
36 M 2803 61 PD
HANDLEY, DENNIS MICHAEL. 8650 W COLLEGE BLVD.66210

649-1311
50 M 2803 76 FP
HARMS, ALBERT C. 5750 WEST 95TH.66207

381-5550
13 M 1902 38 FP
HARPSTER, GENE D. 4121 W 83RD SUITE 216.66208

648-1400
31 M 1902 57 GS
HARTONG, TONY JOSEPH. 8901 W 74TH SUITE 25.66204

362-3210
53 M 1902 78 PD

HARTONG, WILLIAM A. 8901 W 74TH STE 372.66204

831-9300

44 M 1902 71 IM

HATHAWAY, PETER. 11055 CEDAR STE 216.66211

383-2270

31 M 3503 60 IM

HAY, JAMES R. 7720 W 85TH #101.66212

-

58 M 1902

HENRY, JOSEPH E. 8901 W 74TH #348.66204

432-8000

42 M 1902 68 PUD

HERRON, KRISTINE G. 8549 WEDD.66212

-

57 F 1902 84

HESSER, HERBERT H. 7207 EDGEWOOD BLVD.66203

-

06 M 1902 34 DD

HILL, RODNEY W. 8901 W 74TH SUITE 208.66204

362-6161

47 M 1902 74 IM

HITCHCOCK, C THOMAS. 8901 W 74TH SUITE 356.66204

677-2508

47 M GS

HOBSON, MILBURN W. 9119 W 74TH ST.66204

831-2334

30 M 1902 55 D8G

HODES, HERBERT C. 4601 W 109TH #330.66211

381-6868

43 M 1902 69 D8G

HODGES, BRUCE E. 10550 QUIVIRA SUITE 430.66215

888-0777

32 M 1902 63 FP

HODD, ROGER W. 8300 COLLEGE BLVD STE 105.66210

649-9609

49 M 1643 74 JRS

HOPKINS, LENLEY. 8901 W 74TH SUITE 24.66204

722-6121

30 M 3841 56 GS

HOPKINS, WILLIAM O. 8800 W 75TH SUITE 350.66204

831-3500

33 M 2803 61 JRS

HORSEMAN, ROBERT F. 9119 W 74TH ST.66204

432-7419

19 M 1902 44 D8G

HOUSTON II, LAWRENCE MORLEY. 8650 W COLLEGE BLVD.66210

649-1311

50 M 2803 76 FP

HUANG, GEDRGIANA L W. 6300 GLENWOOD #3.66202

384-4998

41 F 4107 75 GP

HUMPHREY, LOREN JENKINS. HUMPHREY MED CL & TUM INS.66204

384-2850

31 M 1611 56 SON

INNES, ROBERT C. 1022 BRIAR.66207

-

25 M 2802 49 DD

ITURRALDE, GEDRGE. 7501 MISSION RD.66208

648-4949

21 M 13201 49 P

JACKSON, ROBERT V. 8901 W 74TH.66204

362-1660

49 M 2803 77 PD

JANES, DONALD R. 10550 QUIVIRA #360.66215

492-1955

34 M 1902 60 D8G

JOHNSON, GARY A. 5106 DUTLDDK.66202

-

55 M 1902 86

JOHNSON, KEITH A. 7630 WINDSOR DR.66208

-

59 M 1902 87

JONES, CHARLES E. SHAWNEE MISSION MED CNTR.66201

676-2214

31 M 1902 60 FP

JONES, DAVID B. 9808 GLENWOOD.66212

-

58 M 1902 84

JONES, H IVDR. 8901 W 74TH SUITE 269.66204

362-4040

24 M 80303 51 P

JOUVENAT, NEIL C. 10550 QUIVIRA SUITE 120.66215

492-1844

43 M 3005 71 D8G

KADIAN, RAJESH S. 10550 QUIVIRA SUITE 260.66215

541-0577

50 M 71 IM

KAGAN, STUART M. 10550 QUIVIRA - SUITE 340.66215

492-1111

44 M 4901 69 PD

KASHYAP, BANSHI PRASAD. 8901 W 74TH SUITE 257.66204

236-4500

47 M 49554 69 IM

KASSER, CHRIS L. 17531 W 70TH.66217

-

53 1902 81 IM

KATZ, FRED S. 8901 W 74TH SUITE 145.66204

722-0020

50 M 1902 79 PS

KEITH, REX B. 9420 MEADOW LANE.66206

-

57 M 1902 85

KELLEY, GORDON R. 4601 W 109TH SUITE 210.66211

341-3040

52 M 6002 77 N

KELLEY, MARSHALL D. 7232 GRANDVIEW DR.66204

-

59 M 1902

KELLY, MICHELE. 6427 W 51ST TERRACE.66202

-

63 F 1902 87

KENNEDY, KENNETH R. 6100 MARTWAY STE 11.66202

432-0126

24 M 1902 53 FP

KETCHUM, LYNN D. 10550 QUIVIRA RD STE 310.66215

492-3737

36 M 2101 60 PS

KHURANA, SATISH K. 9119 W 74TH SUITE G-2.66204

432-3334

41 M 49536 65 PD

KIMURA, CHARLES C. 8901 W 74TH SUITE 125.66204

262-4220

25 M 2101 56 A

KIMURA, STEPHEN H. 3504 W 85TH.66206

-

59 M 1902 86

KODANAZ, A AYTEKIN. 5710 REINHARDT DR.66205

334-2500

28 M 90201 55 ANES

KOZIKOWSKI, BEN M. 7301 MISSION RD.66208

362-8317

30 M 2834 55 DRS

KRISHNAN, LEELA. 8800 NALL AVE.66207

588-3600

43 F 4802 79 RD

KRUEGER, KURT ALLEN. 10002 HOWE DR.66206

648-0323

48 M 3006 74 ANES

KUBIN, DORIS A. 2504 W 71ST.66208

-

15 F 1902 43 DD

KURTH, PAUL H. 5555 W 58TH.66202

432-2080

53 M 2507 77 IM

KURTH, ROBERT H. 5555 W 58TH.66202

432-2080

28 M 3005 53 IM

LAPI, RUTH M. 2012 STRATFORD RD.66208

-

14 F 4107 37 DD

LASH, RAY E. 8901 W 74TH SUITE 21.66204

722-0080

50 M 1902 75 CD

LEATHERS, HOLLISS K. SHAWNEE MISSION MED CNTR.66201

676-2340

38 M 3901 64 PATH

LEAVELL, MICHAEL E. 7102 CODY.66203

-

56 M 1902 84

LEGASPI JR, PEDRO L. SHAWNEE MISSION MED CNTR.66201

676-2479

36 M 74801 60 ANES

LESTER, JOHN BUCKLES. 4140 W 71ST SUITE 108.66208

432-7276

45 M 1902 70 P

LEWIN, WALTER. 8901 W 74TH ST.66204

362-4040

30 M 1902 56 P

LEWIS, JAMES E. 3700 W 83RD SUITE 203.66208

649-0923

37 M 2101 63 P

LIPSEY, JAMES H. 8800 W 75TH SUITE 350.66204

831-3500

31 M 1606 56 DRS

LOKER, JAMES L. 9008 W 82ND TERR.66103

-

56 M 1902 86

LULD, ANTONIO R. 7600 STATE LN.66208

649-3900

35 M 30801 60 IM

LUNBERRY, JULIA J. 6028 EL MONTE.66205

-

55 F 1902 85

MACDUGALL, MARGARET L. 6251 ASH.66202

588-6074

48 F 1901 77 NEP

MADISON, RANDALL W. 7711 GARNETT.66215

52 M 1902 87
MAGEE, SHAWN M. 6105 W 54TH TERR.6620261 M 1902 87
MALLORY, JOHN A. 10550 QUIVIRA SUITE 510.66215
492-6200
43 M 2803 71 IM
MANTZ, FRANK A. 9309 W 103RD.6621212 M 4101 38 DO
MARVIN, NORMAN G. WYCLIFF SHOPPING CENTER.66212
541-8282
29 M 1902 56 FP
MASER, GEORGE R. 5808 NALL.6620212 M 1902 36 DO
MATHEWS, DAVID R. 4601 W 109TH SUITE 212.66211
649-3883
53 M 1902 78 FP
MATHEWS, ROBERT MAJOR. 7301 MISSION RD.66208
362-6888
25 M 1902 54 GS
MATZEN, TED A. 7341 BELINGER.6620852 M 1902 85
MAXWELL, ROBERT A. 8901 W 74TH.66204
362-1660
46 M 1902 73 PD
MCCAUGHEY, HUGH W. 11055 CEDAR SUITE 210.66211
381-1724
28 M 1902 53 IM
MCCOWEN, HERBERT M. 4835 W 62ND TERR.6620258 M 1902
MCDONALD, THOMAS L. 3712 W 48TH.6620553 M 1902 84
MCEACHEN, WILLIAM H. 3700 WEST 83RD SUITE 102.66208
649-3335
32 M 1902 59 PD
MCELROY, MICHAEL B. 6300 GLENWOOD.66202
432-112243 M 2803 69 DRS
MCGURK, THOMAS E. 4601 W 109 SUITE 206.66211
649-2080
39 M 2803 65 P
MCWHERTER, LOTTIE B. 5920 NALL SUITE 308.66202
362-146430 F 1902 57 IE
MENEZ, CESAR V. 4121 W 83RD SUITE 120.66208
381-4484
36 M 74810 56 D
MILLER, FREEMAN LANCE. 10550 QUIVIRA SUITE 340.66215
492-111148 M 1902 74 PD
MISKEW, DON B W. 7301 MISSION RD SUITE 348.66208
362-8317
42 M 6506 69 DRS
MOFFAT, ROBERT E. PO BOX 4250.66204
677-088342 M 1902 68 DR
MORITZ, RICK S. 10806 WEST 98TH.66214
371-4343
54 M 1902 78 ORMORONEY, JEAN M. 10550 QUIVIRA SUITE 510.66215
492-6200
25 F 4107 65 N
MUEHLBERGER, JAMES J. 4601 W 109TH SUITE 314.66211
383-322234 M 3006 60 PD
MUELLER, J KENT. 3700 WEST 83RD SUITE 203.66208
649-0923
35 M 1902 62 D
MUNDEN, FRANK A. 5300 W 94TH TERR.66207
381-467438 M 1902 64 A
MURPHY, JAY W. 8901 W 74TH STE 21.66204
362-6161
49 M 3840 73 CD
MURPHY, TIMOTHY P. 5519 STATE PARK RD.6620553 M 1904 87
MURRAY, W LEE. 4601 W 109TH SUITE 225.66211
381-293135 M 1902 61 DPH
NASH, ROBERT A. 4601 W 109TH STE 206.66211
649-8686
31 M 1902 55 P
NAUER, PAULA LOU. 7301 MISSION RD STE 342.66208
384-0745
49 F 1902 74 FPNAVICKAS, LEONARD A. 8901 W 74TH SUITE 225.66204
362-5510
53 M 1902 77 FP
NEIBURGER, JAMES B. 11111 NALL AVE STE 114.66211
341-890746 M 1642 72 A
NELSON, BRYAN C. 9119 W 74TH SUITE 107.66204
384-5500
59 M 1902 75 PD
NELSON, CHARLES G. 9344 W 49TH.6620356 M 1902 86
NELSON, JOHN B. 10550 QUIVIRA SUITE 510.66215
492-6200
48 M 2846 75 DPH
NIEMAN, JOHN L. PO BOX 7426.66207
381-828228 M 3806 58 PD
NOSTI, JUAN C. 8901 W 74TH SUITE 345.66204
262-5014
38 M 13204 63 PS
NUÑEZ, JULIAN. 10021 MASTIN.6621230 M 27501 60 FP
NYE, C ERIK. 7301 MISSION RD SUITE 348.66208
362-8317
39 M 3520 65 DRS
O'BRYAN, JAMES J. 10550 QUIVIRA.66215
492-252547 M 1902 73 PD
OKTAWIEC, DANUTA. 5848 FONTANA DRIVE.6620522 F 80303 50 ANES
ONG, CATHERINE M. 6001 W 90TH TERR.6620760 F 1902 86
OSGOOD, GEORGE M. 3700 W 83RD.66208
381-5200
14 M 1902 44 GP
OWENS, RICHARD L. 10000 W 75TH SUITE 103.66204
362-368724 M 3006 51 DM
PATTERSON, JOHN R. 5317 CHADWICK RD.6620520 M 1902 48 DO
PEARCE, EUGENE F. J. 9119 W 74TH SUITE 104.66204
722-3102
24 M 2802 49 D8GPEARCE, LUNETTA M. 9119 W 74TH SUITE 104.66204
362-1525
26 F 3005 49 FP
PENTECOST, RICHARD L. 8900 STATE LINE SUITE 350.66206
383-141032 M 1001 56 P
PETELIN, JOSEPH B. 8901 W 74TH SUITE 356.66204
677-2508
49 M 1902 76 GPVSPETERSEN, A GENE. 3700 W 83RD SUITE 104.66208
648-3911
27 M 1902 54 IM
PETERSEN, GERALD D. 3700 WEST 83RD.66208
648-391139 M 1902 60 IM
PETERSEN, MARK I. 4956 ADAMS.66205
722-4185
58 M 1902 84PETIT, CARL ALFONSO. 10550 QUIVIRA SUITE 240.66215
888-1114
33 M 60 GS
PETRIE, SAMUEL C. GEORGETOWN MED BLDG.66202
722-116627 M 1902 58 IM
PFUETZE, BRUCE L. 4601 W 109TH.66211
383-363042 M 1902 68 A
PFUETZE, KARL D. 10550 QUIVIRA SUITE 510.66215
492-6200
40 M 1902 67 CDPHILLIPS, WARREN G. 3700 W 83RD.66208
649-0923
26 M 1902 60 DPILCHARD, WILLIAM A. GEORGETOWN MED BLDG.66204
362-3210
39 M 1602 65 OPHPINGLETON, WILLIAM WARREN. 8901 W 74TH #348.66204
432-8000
42 M 3901 67 PUDPITTS, RONALD L. 8901 W 74TH - SUITE 330.66204
362-2524
35 M 2002 62 DPOONAWALA, HJSENI E. 3001 W 121.66209
561-2025
33 M 49528 59 D

POWELL, CAROL W. 8216 CHEROKEE CIRCLE.66206
391-3785
25 F 1902 51 P
POWELL, KENNETH A. 8216 CHEROKEE CIRCLE.66206
753-7000
25 M 1902 53 IM
PRONKO, MICHAEL J. 4121 W 83RD SUITE 223.66208
648-7878
34 M 1902 60 P
RALSTIN, JAMES H. 14708 W 71ST TERR.66216
299-2069
49 M 1902 74 IM
RAY, LARRY D. 2500 N 105TH.66206

55 M 1902 87
REIVICH, RONALD S. 8900 STATE LN #331.66206
383-3050
34 M 3806 60 P
RICE, BERNARD F. 8901 W 74TH SUITE 125.66204
262-9222
31 M 4113 56 END
RICHTER, DON G. SHAWNEE MISSION MED CTR.66201
676-2464
50 M 1902 79 ANES
RICK JR, GREGORY G. THE GEORGETOWN MED BLDG.66204
831-9300
40 M 1902 66 GE
RIESENMY, BRANDON D. 5538 REEDS.66202

60 M 1902 87
RIFTEL, LAWRENCE D. 10550 QUIVIRA SUITE S10.66215
492-6200
53 M 1902 78 IM
RITCHIE, KAREN S. 8901 W 74TH SUITE 21.66204
722-0080
49 F 3840 73 P
ROBERTSON, EDWARD J. SHAWNEE MISSION MED CTR.66201
676-2479
46 M 1902 78 ANES
ROBINSON, JOHN D. 74TH & GRANDVIEW.66201
676-2479
48 M 1902 74 ANES
ROBINSON, JAMES T. 8800 W 75TH SUITE 310.66204
677-3113
21 M 4812 45 OPH
ROSENBERG, STANTON L. 1900 W 75TH SUITE 200.66208
362-8080
30 M 1902 55 P
ROSS, ALBERT M. 6740 FONTICELLO.66208

59 M 1902 85
RUBIN, HERBERT M. 10550 QUIVIRA - SUITE 340.66215
492-1111
37 M 2803 63 PD
RUMOLD, MERVIN J. 6340 INDIAN LAKE.66208

03 M 1902 30 GS
RYAN, MICHAEL E. 4601 W 109TH SUITE 210.66211
341-4030
46 M 1902 72 V
RYMER, ROBERT A. 8901 W 74TH SUITE 373.66208
722-0170
41 M 0702 68 OPH
SACHEN, FREDERICK L. 9620 RDE.66207

45 M 4813 79 N
SAFFD, KARL S. 8901 W 74TH.66204
362-9505
39 M 52801 62 PS
SATHYANARAYANA, SARASWATHI. 8901 W 74TH SUITE 20.66204
677-2281
45 F 49509 67 OBG
SAWKAR, LAXMIDAS A. 8901 W 74TH SUITE 312.66204
384-4844
36 M 49523 63 ONC
SCHAEFER, JOSEPH PETER. 10550 QUIVIRA - SUITE 230.66215
492-7440
34 M 1902 60 IM
SCHREPFER, ROSEMARY. 6401 ENSLEY LANE.66208
588-6200
22 F 1902 47 OBG
SCHROLL, JOHN T. 8901 W 74TH SUITE 248.66204
384-4990
51 M 1902 76 OBG
SCHUSTER, MICHAEL R. 6427 HALLET.66216

56 M 1902 87
SCIDLARD, CHARLES M. 8822 W 57TH.66202
58 M 1902 84
SCLAR, WILLIAM C. ODCTORS BLDG SUITE 450.66215
492-7730
46 M 2501 72 GS

SCOTT, JUDITH C. 2041 W 84TH TERR.66206
F 1902 86
SEGRAVES, STEVEN D. 8820 SANTA FE LN.66212
59 M 1902 86
SELLERS, JEFF D. 9158 SOMERSET.66207
55 M 1902 86
SHAAD, DOROTHY J. 2322 W 51ST.66205

09 F 1902 44 DO
SHAFFER, KATHLEEN BRAY. 9119 W 74TH SUITE 107.66204
384-5500
54 F 2846 79 PO
SHAFFER, STANLEY G. 9119 W 74TH SUITE 107.66204
384-5500
55 M 2846 79 PO
SHERIDAN, RANDY M. 8901 W 74TH SUITE 36.66204
236-6455
53 M 1902 78 OBG
SHOFSTALL, WILLIAM H. 6100 MARTWAY.66202
11 M 3901 41 JO
SHULTZ, WILLIAM H. 9400 ENSLEY LN.66206

60 M 1902 87
SILLS, THOMAS D. 8309 MULLEN RD.66215
281-8883
49 M 5606 77 EM
SILVER, BRAD J. 8901 W 74TH SUITE 30.66204
432-2280
50 M 1205 76 IM
SMITH, DALE C. 4601 W 109TH ST SUITE 224.66211
381-0353
20 M 1902 45 OPH
SMITH, DONALD J. 8600 W 95TH ST.66212
642-4515
18 M 1902 49 FP
SMITH, WILLIAM P. PO BOX 4250.66204
677-0883
51 M 1902 77 R
SNODGELL, FIRMEN E. 5555 W 58TH.66202
432-2080
31 M 1902 61 IM
SNOW JR, ARTHUR D. 8901 W 74TH - SUITE 225.66204
362-5510
45 M 1902 75 FP
SOELDNER, JAMES J. 8901 W 74TH SUITE 24.66204
722-6214
44 M 1902 70 FP
SPEER, FREDERIC. 5811 OUTLOOK.66202

09 M 1902 34 OO
SPERRY, ROBERT E. 6701 W 87TH APT 101.66212

59 M 1902 86
STAMDS, GEORGE E. 10550 QUIVIRA.66215
492-5456
46 M 1803 72 IM
STEVENSON, E KENT. 4121 W 83RD SUITE 150.66208
649-5566
45 M 2802 67 CMP
STINGO, ANDREW J. 9624 PEIM LN #C.66212

61 M 1902 87
STRIEBINGER, CHARLES M. 4601 W 109TH SUITE 307.66211
341-7299
45 M 1606 67 VS
STUBER, JACK LAWRENCE. PO BOX 4250.66204
677-0883
40 M 1902 66 DR
STUCKEY, CHARLES E. 10550 QUIVIRA SUITE 240.66215
492-7737
41 M 3005 68 GS
SUGAR, ROBERT L. 8901 W 74TH SUITE 248.66204
384-4990
40 M 3508 66 OBG
SULLIVAN, TOM G. 10550 QUIVIRA SUITE 320.66215
492-3600
44 M 1902 71 OBG
SULLIVAN JR, HENRY B. 5817 NIEMAN RD.66203
631-6160
24 M 1902 52 FP
SUTTON JR, RICHARD L. 3203 W 83RD TERR.66206

08 M 2501 29 OO
TAIT, AMY S. 5959 DEARBORN #304.66202
58 F 1902 86
TAIT, LAYNE S. 5959 DEARBORN #304.66202
59 M 1902 86

TALLEY, ROBERT L. 6501 W 101 ST PLACE, 66212
588-3000
48 M 4802 74 IM
TARVIN, RANDY J. 4782 SKYLINE, 66205
-
59 M 1902 86
TAYLOR, THOMAS L. 8901 W 74TH SUITE 34, 66204
362-9444
49 M 1902 66 GS
TENNY, ROBERT T. 4601 W 109TH SUITE 307, 66211
341-7299
51 M 1902 76 NS
TETZLAFF, ARCH D A. 4520 W 65TH, 66208
334-2500
26 M 40721 52 ANES
THEDINGER, BRITT A. 2001 W 49TH TERR, 66205
-
57 M 1902 89
THOMSEN, GARY. 8901 W 74TH SUITE 225, 66204
362-5510
51 M 3005 76 FP
TOLSON, WILLIAM B. 8901 W 74TH SUITE 21, 66204
722-0080
37 M 1902 63 CD
TRETAR, LAWRENCE L. 8901 W 74TH, 66204
677-1776
33 M 1902 60 GS
TROWBRIDGE, DENISE R. 12606 W 110 TER, 66210
-
61 F 1902 87
TUCKER, SHERIDAN G. 4121 W 83RD SUITE 150, 66208
649-5566
59 M 1902 75 CH
VALK, WILLIAM L. 5401 W 81ST, 66208
588-6146
09 M 2501 37 U
VANNAMAN, DONALD D. P D 8DX 7426, 66207
381-8282
43 M 1902 71 PD
VASUNEVAN, GORDON, 5915 HAUSER, 66216
-
60 M 1902 86
VENNEMAN II, CHARLES R. 10505 W 89TH, 66214
-
53 M 1902
VILE, SHELDON B. 8341 BRIAR, 66207
-
61 M 1902 87
WALL, PHIL, LEONARD L. 5011 NEDSAD AVE, 66205
384-3342
50 M 1902
WANG, SIDNEY W. 10550 QUIVIRA SUITE 130, 66215
492-1500
32 M 38503 58 FP
WATANABE, MASAYO, 5801 W 92ND TERR, 66207
-
50 M 1902 86
WAXMAN, DAVID. 12516 W 85TH TERR, 66215
888-7889
18 M 3515 50 IM
WEBB, JAMES R. 5949 NIEMAN ROAD, 66203
631-0900
34 M 1902 61 FP
WEINGART, JAMES H. 4717 W 70TH, 66208
-
58 M 1902 84
WELLING, PAUL A. 6105 W 54 TERR, 66202
-
61 M 1902 87
WERTH, CLAUDE J. 4121 W 83RD, 66208
649-5566
37 M 1902 64 P
WHITEHEAD, RICHARD E. 7301 MISSION RD, 66208
362-8317
31 M 2501 58 JRS
WHITLEY, DOUGLAS M. 4601 W 109TH SUITE 202, 66211
341-4770
34 M 1902 60 D
WIGGINTON, GERALD D. D.D., 9119 W 74TH SUITE 107, 66204
384-5500
44 M 2878 70 PD
WILEY, JOHN H. 9119 W 74TH SUITE 201, 66204
831-2334
37 M 4113 63 D8G
WILLIAMS, THOMAS A. 9601 MANDR ROAD, 66206
577-5152
36 M 1902 62 FP
WILSON, ROBERT B. 6915 W 51ST PLACE, 66202
-
10 M 1902 40 DD
WILSON, SLDAN J. 5618 W 62ND, 66202
-
10 M 1902 36 HEM

WDDDS, DENNIS D. 5435 BRIAR, 66205
-
60 M 1902
WDDDS, GREGORY A. 5300 W 49TH, 66205
-
56 M 1902 83 DRS
WDDDS, MICHAEL S. 7601 GARNETT #10, 66214
-
61 M 1902 87
WRIGHT, CHRISTOPHER. 4624 W 62ND, 66205
-
61 M 1902 87
WULSER, MICHAEL J. 13207 W 94TH, 66215
-
M 1902 87
WURSTER, GEDRGE R. 3700 W 83RD SUITE 203, 66208
649-0923
35 M 1902 61 P
YE, RICHARD C. 7301 MISSION RD, 66208
362-7505
20 M 24222 46 PS
YOHE, RUTH M. 8600 W 95TH ST, 66212
383-3377
26 F 4107 54 PDA
YDUNG, JOHN W. 8220 TRAVIS STE 115, 66204
383-1550
37 M 4706 63 PS
ZACK, ASHLEY S. 4601 WEST 109TH SUITE 122, 66211
642-4040
46 M 2803 73 PD
ZAMIEROWSKI, DAVID S. 8800 W 75TH SUITE 340, 66204
831-4113
42 M 2307 68 PS
ZIMMERMAN, DANIEL D. 4761 RAINBOW, 66205
831-0910
45 M 3005 70 IM

SMITH CENTER—913
(Central Kansas Society)

SHEPPARD, ROBERT G. 120 E COURT, 66967
282-6654
21 M 1902 45 GS
STEINKRUGER, VERLYN WILLIAM. 120 E COURT, 66967
282-6654
29 M 3005 53 FP
WDDDS, HUGH J. 120 EAST COURT, 66967
282-6654
26 M 1902 52 FP

SOUTH HAVEN—316
(Tri-County Society)

USBLAKER, ERNEST J. , 67140
892-2261
11 M 1902 38 FP

SPEARVILLE—316
(Barton County Society)

QUACKENBUSH, ROBERT P. SPEARVILLE DIST HDSP, 67876
549-3471
21 M 1611 52 FP

ST. FRANCIS—913
(Northwest Kansas Society)

CRAM, ERNEST R. PD 8DX 625, 67756
332-2126
24 M 1902 52 FP
JEWELL, ROSS L. 203 E SPENCER, 67756
332-2832
13 M 1902 56 FP
STEPHENSON, LUCILLE C. , 67756
-
06 F 1902 32 FP
WALZ, THOMAS J. 115 SOUTH QUINCY, 67756
-
94 M 1902 21 DD

ST. MARYS—913
(Pottawatomie County Society)

BROWN, FRED E. 602 W PALMER, 66536
437-2256
26 M 1902 SS FP

STAFFORD — 316
(Ninnescah Society)

BROWN, C EVERETT, 102 N MAIN, 67578
234-5251
10 M 1902 47 A
QUIJAND JR, RAMON S, 412 E GRAN, 67578
234-5236
45 M 74811 71 GP

STERLING—316
(Rice County Society)

OYSART, JACK C, 224 N FOURTH, 67579
278-2181
12 M 3901 39 JO
SIMPSON, TOM C, 239 N BROADWAY, 67579
278-2123
47 M 1902 73 FP

STOCKTON—913
(Central Kansas Society)

MAUCK, HAROLD C, 623 SOUTH 2ND, 67669
425-6280
20 M 1902 54 FP
VDTAPKA, WILLIAM L, 623 S SECOND, 67669
425-6280
24 M 1902 53 FP

SUBLETTE—316
(Southwest Kansas Society)

THIEMANN, A H, D.O., 301 DERBY, 67877
675-2241
12 M 1805 42 FP

SYRACUSE—316
(Southwest Kansas Society)

PETTERSON, CECIL E, PROFESSIONAL ASSOCIATION, 67878
384-5731
14 M 1902 39 FP

TONGANOXIE—913
(Leavenworth County Society)

STEVENS, PHILIP L, BOX 319, 66086
845-2090
27 M 1902 54 FP

TOPEKA—913
(Shawnee County Society)

AGAN, LAWRENCE M, 1314 PEMBROKE LN, 66604
357-4306
20 M 5002 44 R
ARJUNAN, K N, 901 GARFIELD, 66606
357-6171
44 M 49568 73 NS
ARREDONDO, MARIO, 6024 SW 26TH, 66614
296-7216
25 M 26401 54 P
ARUNAKUL, PUNYA, 1710 W TENTH, 66604
234-2624
44 M 89104 69 JTD
ASHLEY, BYRON J, 3222 PLASS, 66611
233-2280
98 M 1902 24 OPH
ASHLEY JR, B JOHN, 1616 WEST 8TH ST, 66606
233-2280
31 M 1902 56 OPH

AVERILL, STUART C, MENNINGER FD, 66601
273-7500
24 M 502 52 P
BAHR, RALPH H, S-F CAP REG RADIDTHER CTR, 66606
295-8008
35 M 1606 59 R
BAI, EUNG KOD, 631 HORNE SUITE 110, 66606
232-6964
46 M 70 FP
BAIR, GLENN O, 634 MULVANE SUITE 400, 66606
233-5153
31 M 2401 57 1M
BAKER, FREDERICK C, 2101 WEST 10TH, 66604
232-0909
35 M 4113 62 FP
BAKER, PHILLIP L, 909 MULVANE, 66606
357-0301
37 M 3005 63 ORS
BAKER, RAY O, 1615 W EIGHTH, 66601
233-8961
30 M 4812 55 GPM
BARABAN, MARC R, 1319 HUNTDON, 66604
357-5325
50 M 2846 75 PS
BASSETT, P MARCUS, 2511 SW WESTPORT DR, 66614
742-7915
51 M 1902 77 FP
BAUCDM, KARAN YVONNE, 1516 W SIXTH, 66606
233-1639
50 F 1902 75 DBG
BAUCDM-COPELAND, SHARDN LAVARNE, 634 MULVANE STE 402, 66606
235-9544
50 F 2846 75 FP
BEACH, RICHARD R, 106 MED ARTS BLDG WEST, 66604
233-7943
23 M 2802 48 1M
BECK, JOSEPH O, 2760 SW BURLINGAME, 66611
-
13 M 3005 43 JO
BEDFORD, D R, PO BOX 1772, 66615
-
09 M 4802 40 1M
BEELMAN, FLOYD C, 1286 LAKESIDE DR, 66604
-
02 M 3840 35 FP
BELLER, WILLIS L, 31 PEPPERTREE LANE, 66611
-
14 M 1902 41 DO
BENSON, DAVID R, 6631 SW 40TH, 66610
-
51 M 1902 87
BLAKE, HENRY S, 1933 WESTWOOD DR, 66604
-
11 M 3520 37 DO
BONEBRAKE, C RICHARD, 634 MULVANE STE 104, 66606
233-1979
48 M 1606 75 DBG
BOREL, DAVID, ST FRANCIS HOSP & MED CTR, 66606
295-8473
45 M 1902 71 PATH
BDWEN, CLDYIS W, 2200 WEST 10TH, 66604
234-8601
12 M 1902 37 FP
BDWEN JR, HARRY J, 2200 WEST 10TH, 66604
234-8601
11 M 1902 37 FP
BOYO, SPENCER H, 1815 WEST 2ND, 66606
-
11 M 1902 35 DBG
BRAHMAN, HERBERT D, 1700 SEVENTH, 66606
295-8471
43 M 512 70 PATH
BRAUN, ROBERT W, 901 GARFIELD, 66606
354-9591
44 M 2803 70 1M
BRAUNSDORF, ROBERT L, 2843 VIRGINIA, 66605
-
08 M 2501 35 FP
BRIDWELL, RUSSELL E, 1710 W TENTH, 66604
234-2624
26 M 1902 51 ENT
BRUSCHI, WALTER C, 4511 W 33RD TERR, 66614
682-2000
24 M 6702 53 P
BUROZIK, EBERHARD G, 2700 W SIXTH, 66606
296-4222
26 M 40916 54 P
BUSKIRK, STEVEN J, 310 MED ARTS BLDG, 66604
234-3451
54 M 3005 79 TR
BYLANDER, TERESA L, D.O., 1530 BURNETT, 66604
-
46 F 1875 81 P

CACHIA, RICHARD M, DEPT OF PATHOLOGY,66606
295-8472

51 M 62701 73 PATH
CASHMAN JR, MAJRICE R, 901 GARFIELD,66606
354-9591

35 M 1902 61 HEM
CAVANAUGH, JOHN W, 200 PROFESSIONAL BLDG,66604
235-3488

13 M 1803 39 GS
CHAMBERLIN JR, CECIL R, BOX 829,66601
273-7500

30 M 3901 55 CHP
CHEN, CHU-CHI, 918 W TENTH,66604
354-4465

47 M 24405 73 U
CHEN, TAK-MING, 308 MED ARTS BLDG,66604
234-3451

41 M 24402 68 ANES
CHERRY JR, ARTHUR C, 918 WEST 10TH,66604
233-3362

27 M 3806 53 PD
CLARK, CRAIG N, 3124 E 6TH,66607
354-7683

23 M 1902 58 FP
COCHRAN, PAUL W, MENNINGER FD,66601
273-7500

33 M 4802 58 IM
COHEN, LOUIS, 918 W 10TH,66604
233-7175

14 M 1902 41 IM
COKELEY, JOHN M, 2200 GAGE BOULEVARD,66622
272-3111

30 M 5104 55 D
COKER, W LAURENCE, 631 HORNE #340,66605
232-9394

53 M 1902 78 FP
COLLINS, DEAN T, MENNINGER FD,66601
273-7500

28 M 1902 55 D
COLLINS, EDWARD JOSEPH, 900 WASHBURN,66606
233-3242

45 M 1611 71 OPH
COLLINS, ELISABETH B, 634 MULVANE #402,66606
233-6399

21 F 40905 46 D
COLLINS, FRANCIS T, 206 MED ARTS BLDG EAST,66604
233-6470

14 M 1902 43 IM
CONROY, ROBERT W, MENNINGER FD,66601
273-7500

38 M 2604 64 D
COOLEY, DENNIS M, TOPEKA MEDICAL CENTER,66604
233-3362

51 M 1902 77 PD
COTTON, ROBERT T, 901 GARFIELD,66606
354-9591

19 M 1902 45 IM
CRARY, JOHN E, 201 MED PLAZA BLDG,66604
233-4202

18 M 1902 43 IM
CROUCH, STEVEN W, 904 MULVANE,66606
232-8224

51 M 1902 76 PD
CROUCH, WILLIAM H, 904 MULVANE,66606
232-8224

20 M 2802 45 PD
DAVIS, CHESTER R, 631 HORNE,66606
232-6020

50 M 1902 75 FP
DECORE, ROMAND, BOX 829,66601
273-7500

30 M 64901 50 D
DELGAUD, SERGIO, 634 MULVANE STE 200,66606
357-0352

37 M 2501 62 ORS
DESJIGNIE, RAFAEL R, 3107 W 21ST,66604
296-5306

19 M 27501 D
DUNAGIN, JACK A, 925 WESTERN,66606
233-7138

20 M 1902 44 P
DUNIVEN, PHILIP L, RADIOLOGY & NUCLEAR MED,66604
234-3451

52 M 4812 77 R
DURST JR, ROBERT D, CONTINENTAL MED BLDG,66606
357-5166

42 M 2803 69 D
EATON, EDWARD L, 925 WESTERN,66606
233-7138

40 M 0401 73 P
ELDER, DOUGLAS M, 310 MED ARTS BLDG,66604
234-3451

41 M 1902 69 DR
FAIRCHILD, RICHARD S, 901 GARFIELD,66606
354-9591

48 M 1902 76 IM
FEAGAN, JERRY, 901 GARFIELD,66606
354-9591

39 M 1902 63 GE
FERNANDEZ, LUIS A, 2707 WEST 13TH,66604
233-8961

14 M 27501 41 PD
FIELD, RICHARD A, 308 MED ARTS BLDG,66604
235-3451

29 M 1902 55 ANES
FILLMAN, ELDON M, 301 MEDICAL PLAZA BLDG,66604
233-4256

23 M 1611 44 U
FORD, FRED L, 1264 LAKESIDE DR,66604
-

11 M 2501 36 GS
FOSTER, J BERNARD, 3111 JEWELL,66611
272-3111

14 M 2501 38 PD
FRANKLIN JR, BENJAMIN A, 1001 HORNE #310,66604
234-3451

45 M 1902 76 R
GANDHI, SHANTIKUMAR K, 634 SW MULVANE SUITE 203,66604
233-1690

40 M 49501 67 TS
GAYZARAIN, RAMON C, MENNINGER FD,66601
273-7500

23 M 23101 47 D
GAY, JOHN D, 310 MED ARTS BLDG,66604
234-3451

42 M 4802 68 DR
GENDEL, JOSEPH E, 918 W 10TH,66604
235-9914

12 M 4804 37 ORS
GIESSEL, MICHAEL D, CONTINENTAL MED BLDG,66606
357-5166

48 M 1902 74 D
GIMPLE, KENNETH, 631 HORNE #200,66606
233-7491

45 M 1902 71 ORS
GLEASON, JIMMIE A, 800 LINCOLN,66606
233-5101

33 M 1902 58 DRG
GODFREY, KENNETH E, 9550 SW 53RD,66604
272-3111

19 M 3901 50 P
GRAHAM JR, CHARLES P, 631 HORNE #400,66606
354-9504

40 M 3601 65 GS
GRAY, DAVID E, 3633 SW DRURY LN, 66604
-

16 M 1606 42 GEN
GRAYB, ANTOINE S, 1625 JAKLEY,66604
354-5100

18 M 60501 46 IM
GREENBERG, MARK, 310 MED ARTS BLDG,66604
234-3451

46 M 1611 72 R
GREENE, HORACE T, 1710 W 10TH,66604
354-7508

15 M 401 42 FP
GREENE, RUSSELL E, 5617 FOXCROFT CIRCLE N,66614
295-8008

53 M 515 79 RT
GREENWOOD, EDWARD D, 1915 WEBSTER,66604
-

01 M 702 38 CHP
GREER, RICHARD M, 918 W 10TH,66604
233-5084

09 M 1902 39 DD
GUTOVITZ, ALLEN LOUIS, 634 MULVANE SUITE 100,66606
233-9643

46 M 1611 72 CD
HACKER, ELAINE MARY, 3026 QUAIL CREEK,66614
296-3981

25 F 2604 50 DRG
HALLEY, M MARTIN, 40 MED ARTS BLDG,66604
233-1710

27 M 2401 53 TS
HARRIS, HUBERT L, 210 MED ARTS BLDG,66604
233-3151

12 M 1803 39 D
HARRIS, PATRICIA A, 1617 W 26TH,66611
354-1906

29 F 1902 54 IM
HARRISON, HALL E, 901 GARFIELD,66606
354-9591

39 M 2802 65 IM
HARVEY, R CLAY, 1001 HORNE #310,66604
234-3451

52 M 1902 78 R

HEBBAR, SATYA N. 634 MULVANE SUITE 100.66606
233-9643
39 M 49509 62 CO
HERRERA, JORGE J. 2825 CALIFORNIA.66605
267-5370
27 M 64901 55 IM
HICKMAN, JAMES STEPHEN. 634 MULVANE SUITE 103.66606
233-8508
53 M 2803 79 PD
HIEBERT, JOHN S. 901 GARFIELD.66606
354-9591
40 M 1902 68 CO
HILL, ROBERT N. 901 GARFIELD.66606
354-9591
14 M 1902 67 IM
HILST, WILBUR D. 1271 WOODHULL.66604
273-5870
31 M 3005 55 GS
HIRSCHBERG, J OTTER, MENNINGER FO.66601
273-7500
15 M 1602 40 CHP
HISZCZYNSKYJ, ROMAN. 1500 W TENTH.66604
354-6870
35 M 1803 66 PATH
HOBBS, DONALD D. 200 CONTINENTAL MED BLDG.66606
233-7491
28 M 2401 54 ORS
HOMERZ, DAVID G. 631 HORNE SUITE 220.66606
235-1170
45 M 1803 72 TS
HOLMES, ROBERT W. 901 GARFIELD.66606
354-9591
52 M 1902 74 IM
HORNE, JAMES B. 1305 LAKESIDE DR.66604
272-3111
26 M 1606 52 P
HOSTETTER, JAMES P. 1500 W TENTH.66606
354-6100
43 M 1902 69 EM
HOYT, ARTHUR W. 2521 NW 35TH.66618
234-5663
14 M 2501 40 P
HSU, CHENG H. 1516 W SIXTH.66606
232-1005
41 M 38502 66 U
HSU, SHIN-FU. MEDICAL PLAZA BLDG.66604
232-0362
43 M 24402 68 OTO
HUANG, JONSON. 901 GARFIELD.66606
357-6171
52 M 2701 77 N
HUSTON, JOSEPH W. 634 MULVANE.66606
357-0352
35 M 1902 62 ORS
HUTTON, FREDERICK A. 102 MED PLAZA BLDG.66604
234-0553
29 M 6701 58 PS
HYLAND, JOSEPH M. 80X 829.66601
273-7500
45 M 53902 68 P
ILIFF, R DOUGLAS. 1500 SW JAKLEY.66604
354-6100
49 M 1902 74 FP
ILORETA, ALFREDO T. 1516 W SIXTH.66606
232-1005
47 M 74801 71 U
ISAACSON, RICHARD N. 1001 GARFIELD STE 301.66604
233-4256
48 M 2501 75 U
JACKSON, LINDA H. 212 WOODLAWN AVE.66606
273-7500
42 F 3601 67 CHP
JACOBY II, ROBERT E. 340 CONT MED BLDG.66606
232-9394
46 M 2307 72 FP
JANSSEN, ERWIN T. MENNINGER FO.66601
273-7500
35 M 1803 62 P
JOSEPH, BRIAN W. 3600 SW BURLINGAME.66611
267-0150
38 M 35205 61 CHP
JOSS, CHARLES S. 221 MED ARTS BLDG W.66604
232-0444
14 M 1606 40 GS
JOYCE, G BERNARD. 4929 WEST HILLS DR.66606
17 M 1902 44 OO
KATZ, JEROME B. 80X 829.66601
273-7500
22 M 2101 44 P
KAVEL, KARL K. MED PLAZA BLDG.66604
234-2663
36 M 3605 64 POA

KEARNS, NORBERT W. 80X 829.66601
273-7500
43 M 1002 70 P
KELLY, DAN A. 904 MULVANE.66606
232-8224
39 M 2803 64 PD
KEYS JR, ROBERT C. 308 MED ARTS BLDG.66604
235-3451
36 M 1902 62 ANES
KEYS SR. ROBERT C. 3034 LYDIA.66614
37 M 1902 27 OO
KIM, YONG W. CONT MED BLDG.66606
232-6964
28 M 58302 49 IM
KINDLING, PAUL H. 40 MED ARTS BLDG.66604
233-1710
30 M 3545 61 TS
KIRKEGAARD, RODGER S. 835 WESTERN.66606
233-6493
30 M 1803 56 OPH
KLEINHOLZ JR, EMIL JOHN. 634 MULVANE APT 201.66606
232-1227
39 M 3503 65 IM
KLEMMER, HERBERT. 904 MULVANE MED PARK CL.66606
233-5033
11 M 4102 37 P
KOONTZ, JUDITH A. 904 MULVANE MED PARK CL.66606
233-5033
49 F 1902 75 CHP
KOVARIK, ERNEST D. 900 WASHBURN.66606
233-1800
36 M 3005 64 OPH
KOWALSKI, STEPHEN F. 1615 MEADOWS AVE.66604
273-7500
55 M 3901 81 P
KROLL, HARRY G. 200 CONTINENTAL MED BLDG.66606
233-7491
24 M 1602 50 ORS
LACCHEO, MICHAEL L. 2115 W 10TH.66604
354-4600
51 M 3840 76 FP
LAI, MAX G. 918 W TENTH.66604
354-4465
45 M 24405 72 U
LAI-CHEN, SHEUE-HUEY. 918 W TENTH.66604
354-4465
48 F 38505 73 GP
LAUVEY, WALTON S. 1001 HORNE #310.66608
234-3451
39 M 4804 75 R
LAWSON, OWIGHT. 1203 SW 29TH.66611
06 M 2802 30 IM
LEE, CATHERINE. 600 MADISON.66607
354-5224
49 F 2307 75 IM
LEE, SONG OOW. 308 MED ARTS BLDG.66604
235-3451
43 M 38505 68 ANES
LEE, SONG PING. 918 W 10TH.66604
233-6001
34 M 38502 61 OTO
LEGLER, GARY LEE, D.O.. 2835 SE SKYVIEW CT.66605
233-8961
50 M 2878 77 GP
LEIFER, WILLIAM W. 1500 W TENTH.66606
354-6870
47 M 1902 73 PATH
LENTZ, WILLIAM R. 300 MEDICAL ARTS BLDG.66604
235-3443
24 M 1902 53 FP
LESSENDOEN JR, C M. 5535 NW BRICKYARD RD.66618
272-3111
18 M 1902 43 O
LEVY, EDWIN Z. 4125 SW GAGE CTR DR L-#6.66604
273-5610
29 M 1606 54 P
LIESMANN, GEORGE E. 631 HORNE SUITE 220.66606
235-1170
49 M 1902 74 GPVS
LIESMANN, JEAN ELIZABETH. MEMORIAL HOSP.66607
354-5164
49 F 1902 74 IM
LOONEY, GERALD L. MEMORIAL HOSP.66607
354-5100
37 M 2307 63 EM
LUI, NASON. 1516 W SIXTH.66606
233-1747
49 M 1606 77 GPVS
LUMB, RAYMOND C. 901 GARFIELD.66606
354-9591
42 M 1001 68 RLU

LYNCH, JOHN A. 909 MULVANE,66606

357-0301

30 M 2834 55 ORS

MARSHALL, B M. 1826 SW 34TH,66611

08 M 2802 34 U

MARTIN, WILLIAM D. 308 MED ARTS BLDG,66604

235-3451

19 M 1902 44 ANES

MARTINAK, JOSEPH F. 5144 SW VORSE RD,66614

39 M 3506 66 EM

MAU, WALTER. 301 MEDICAL PLAZA BLDG,66604

233-4256

16 M 1611 40 U

MCCARTER, QUANE K. 2101 W 10TH,66604

233-8979

26 M 1902 58 IM

MCCLELLAN, JOHN W. 2701 BOSWELL,66611

11 M 3006 36 OO

MCCURE, JAMES A. 1541 WESTOVER RD,66604

18 M 1902 44 OO

MCCOMAS JR. MARMADUKE D. 3020 BRUSH CREEK CIRCLE,66614

16 M 1902 43 OO

MCCOY, MICHAEL T. 909 MULVANE,66606

357-0301

49 M 1902 75 ORS

MCELROY, ROBERT T. 221 MED ARTS BLDG,66604

232-0444

35 M 1902 61 GS

MCELROY, WILBJR J. 1616 W EIGHTH,66606

233-2280

35 M 1902 61 JPH

MECH, ARNO D W. 3240 TIMBERLAKE LN,66614

273-7500

52 M 1643 77 P

MEGIBOW, ALAN D. 925 WESTERN,66606

233-7138

37 M 3503 64 CHP

MEIDINGER, RICHARD. 310 MED ARTS BLDG,66604

295-8011

39 M 1902 65 OR

MENNINGER, KARL A. BOX 829,66601

273-7500

93 M 2401 17 P

MENNINGER, ROBERT G. 234 KANSAS,66603

232-7214

22 M 3545 52 P

MENNINGER, ROY W. BOX 829,66601

273-7500

26 M 3520 51 P

MENNINGER, W WALTER, THE MENNINGER FD,66601

273-7500

31 M 3520 57 P

MEYER, MARK E. 3309 SW STONE,66614

577-4818

53 M 1902

MEYER, D WARREN. 634 MULVANE #100,66605

233-9643

49 M 1902 74 CO

MILLS JR, PHILIP E. 901 GARFIELD,66606

357-6171

36 M 1902 64 M

MODLIN, HERBERT C. MENNINGER FD,66601

273-7500

13 M 3005 38 P

MOORE, HUGH C. 1500 W TENTH,66606

354-6870

33 M 4812 59 PATH

MORRIS, MERLE D. 1401 W 10TH,66604

234-2877

21 M 1902 45 JBG

MORRISON, MICHAEL R. 800 LINCOLN,66606

233-5101

50 M 1902 76 JBG

MORROW JR. J TARLTON. 235 WOODLAWN,66606

23 M 4804 47 OO

MUELLER, ARNOLD V. 901 GARFIELD,66606

354-9591

31 M 3005 57 IM

MYERS, JO ANN. MENNINGER FD,66601

273-7500

28 F 1902 53 P

NABOURS, RICHARD D. 4228 W 29TH ST TERR,66614

272-7190

27 M 1902 54 FP

NANCE, JOEL. MENNINGER FOUNDATION,66601

273-7500

42 M 3546 72 P

NATHAN, WILLIAM A. 904 MULVANE,66606

233-5033

48 M 3503 72 CHP

NICE, G WILLIAM. 112 MED ARTS BLDG EAST,66604

235-8090

22 M 1902 46 IM

NICHOLS, JEFF S. 1412 W SIXTH,66606

232-6950

44 M 1902 75 PYM

NOVOTNY, PETER C. MENNINGER FD,66601

273-7500

30 M 15407 55 P

O'NEIL, ROBERT H. 901 GARFIELD,66606

354-9591

20 M 1902 45 IM

O'BURN, ROBERT L. 1150 OAKLEY,66604

273-7500

19 M 2802 50 P

OWEN III, JAMES W. 3457 JARDINE TERR,66611

234-3451

54 M 2802 79 OR

PALMBERG, KENT E. 901 GARFIELD,66606

354-9591

49 M 1902 74 IM

PARMAN, ROBERT D. 904 MULVANE,66606

232-8224

27 M 1902 54 PO

PATEL, VINOD. 655 WESTCHESTER RD,66601

354-5174

47 M 49531 69 V

PATRICK, FRED EDWARD. 904 MULVANE,66606

232-8224

45 M 1902 71 PO

PAYNE, ROBERT R. 200 CONTINENTAL MED BLDG,66606

233-7491

29 M 1902 55 ORS

PENN, GEORGE M. VETERANS MEDICAL CENTER,66622

272-3111

30 M 4802 58 P

PEROVE II, W LANG. 631 HORNE #400,66606

354-9504

49 M 1902 74 GS

PETERSON, DEAN L. 308 MED ARTS BLDG WEST,66604

235-3451

24 M 1902 54 ANES

PETERSON, ROBERT L. STORMONT-VAIL EMERGENCY,66606

354-6108

36 M 1902 62 EM

PETERSON, VERNON J. 310 MED ARTS BLDG,66604

234-3451

42 M 512 68 R

PETRIK, EDWIN L. MEMORIAL HOSPITAL,66607

354-5164

35 M 1902 64 IM

PETTERSON, DENNIS CRAIG. 310 MEDICAL ARTS BLDG,66604

234-3451

49 M 1902 74 R

PFUTZE, ROBERT E. 209 MED ARTS BLDG EAST,66604

232-3332

09 M 1902 35 OBG

PIERCE, CHARLES F. 918 W 10TH,66604

235-6282

24 M 4101 51 OBG

PIERCE, OYALO R. 307 MED ARTS BLDG EAST,66604

235-2226

23 M 5101 49 FP

POLLY, RICHARD E. 909 MULVANE,66606

357-0301

42 M 1803 68 JRS

PORTER, ROBERT D. 901 GARFIELD,66606

354-9591

41 M 2802 67 IM

POWELL, WILLIAM R. 833 GARFIELD,66606

233-8941

30 M 1902 54 GS

POWELL II, BENSON M. 400 CONTINENTAL MED BLDG,66606

354-9504

26 M 1606 49 TS

PRESTON, RALPH R. 1710 WEST 10TH,66604

234-2624

19 M 1902 44 OPH

PROCHASKA, MARK L. 4134 W 6TH APT 314,66606

57 M 1902

PROKOP, BRADFORD S. 900 WASHBURN,66604

233-3900

32 M 1606 57 OPH

PYLE, LUCIEN R. 3139 CANTERBURY LN,66604

01 M 1601 28 OBG

RAINBOW-EARHART, KATHRYN A. 2916 KENTUCKY,66605

233-1730

21 F 4707 48 P

RAJJ, A S PADMA, 918 W 10TH,66604
 234-3211
 39 M 49509 61 TS
 RAMSEY, BARTLETT W, 904 MULVANE,66606
 232-8224
 25 M 1902 50 PD
 RAMSEY, GRACE A, 800 LINDOLN,66606
 233-5105
 48 F 1902 80 D8G
 RANDALL, GORDON R, 1001 HORNE SUITE 310,66604
 234-3451
 50 M 4706 78 R
 RANDELL, EDGAR C, 800 LINDOLN,66606
 233-5101
 41 M 3005 66 D8G
 RANSOM, JAMES H, MEDICAL PLAZA BLDG,66604
 234-2663
 36 M 1803 62 A
 REINKING, VICTOR E, 918 WEST 10TH,66604
 233-5084
 26 M 1902 51 IM
 REYMOND, RALPH D, S-F CAP REG RADIOOTHER CTR,66606
 234-3451
 37 M 2301 67 R
 RICCI, ROBERT LAWLER, 901 GARFIELD,66606
 354-9591
 50 M 1902 75 IM
 RICH, JOSEPH E, WOMEN'S HEALTH CTR,66606
 233-2700
 47 M 40921 74 D8G
 RICHARDS, J M, 634 SW MULVANE #404,66606
 232-7762
 47 M 1611 74 IM
 ROBERTS, WARREN E, PD 8DX 4047,66604
 272-5797
 25 M 1902 57 FP
 ROBINSON, DAVID B, 800 LINDOLN,66606
 233-5101
 47 M 1902 73 D8G
 ROEDER, ROBERT E, 901 GARFIELD,66606
 354-9591
 40 M 1902 67 IM
 RDEMBACH, JEANINE L, 8DX 829,66601
 273-7500
 52 F 1902 75 CHP
 RDSS, JACK L, MENNINGER FD,66601
 273-7500
 32 M 4812 56 P
 RDTERT, LARRY, 301 MEDICAL PLAZA BLDG,66604
 233-4256
 38 M 3005 66 U
 ROY, WILLIAM R, 634 MULVANE STE 104,66605
 233-1979
 26 M 1606 48 ADM
 RUNNELS, JOHN B, 901 GARFIELD,66606
 357-6171
 35 M 1902 61 NS
 RUPP, RICHARD J, 901 GARFIELD,66606
 354-9591
 42 M 3841 68 CD
 SAMPAT, PRAVIN, 2707 WEST 29TH,66614
 272-8440
 51 M 49596 74 IM
 SANCHEZ, ROGELIO, 1516 W 6TH ST,66606
 232-1005
 31 M 64901 64 U
 SARGENT, JOSEPH D, MENNINGER FD,66601
 273-7500
 32 M 2501 58 IM
 SAYLOR, EDWARD H, 918 WEST 10TH,66604
 233-3362
 39 M 1902 65 PD
 SAYLOR, LESLIE L, 918 WEST 10TH,66604
 -
 07 M 1606 35 DD
 SAYLOR, MARK, 918 WEST 10TH,66604
 234-3211
 37 M 1902 66 GS
 SAYLOR, STEPHEN, 631 HORNE SUITE 340,66606
 232-9394
 47 M 1902 73 FP
 SCAMMAN, W WILKE, PD 8DX C-50,66601
 234-8621
 32 M 4705 57 PATH
 SCHLESSEER, HARVEY L, 918 MERCHANTS NATL BK,66612
 235-3184
 21 M 3901 51 P
 SCHLESSEER, PATRICIA T, 1914 WARNER CT,66604
 962-9360
 24 F 3901 49 PD
 SCHRAM, PETER CHARLES, PD 8DX 2428,66601
 273-7500
 39 M 2507 69 P

SEGERS, JOHN A, 901 GARFIELD,66606
 357-6171
 18 M 3545 43 N
 SEHDEV, JIAN, 1001 HORNE SUITE 303,66604
 233-3553
 40 F 6101 63 FP
 SETTLE SR, RUSSELL D, 3019 MAUPIN LN UNIT 101,66614
 -
 04 M 1902 29 DD
 SHAW, JOSEPH L, MED ARTS BLDG #204,66604
 235-6221
 34 M 511 60 DRS
 SHEAFER, DOUGLAS, 925 WESTERN,66606
 233-7138
 34 M 1902 60 P
 SHELTON, STEPHEN E, 925 WESTERN,66606
 233-7138
 35 M 702 61 P
 SHERWOOD JR, CLARENCE E, CONTINENTAL MED BLDG,66606
 354-9504
 22 M 702 53 GS
 SHEU, W ERIC, 1001 HORNE SUITE 308,66604
 235-3451
 43 M 38505 67 ANES
 SIMPSON, WILLIAM S, MENNINGER FOUNDATION,66601
 273-7500
 24 M 6001 48 P
 SISK, PHILLIP B, 310 MED ARTS BLDG WEST,66604
 234-3451
 32 M 1803 56 R
 SMITH, LEO A, 4240 EMLAND DR #8 FDMTBLU,66606
 -
 08 M 3006 33 DD
 SNARR, JACK W, MED ARTS BLDG W #310,66604
 234-3451
 41 M 6201 65 JR
 SDHN, YUNGGYD, 6130 SW 26TH,66614
 232-8761
 40 M 58304 66 ANES
 SPEARMAN, JESSE L, MED ARTS BLDG ROOM 24,66604
 234-2879
 20 M 1902 54 D8G
 SPENCER, MILLARD C, 310 MED ARTS BUILDING,66604
 234-3451
 28 M 1902 55 R
 SPENCER, WAYNE E, 103 MED ARTS BLDG EAST,66604
 233-9686
 38 M 1902 64 GE
 STEIN, JOSEPH M, 901 GARFIELD,66606
 357-6171
 24 M 3519 47 V
 STOCK, KARL W, 2740 BURLINGAME RD,66611
 -
 13 M 2834 37 DD
 SUFI, M ASHRAF, 634 MULVANE #201A,66604
 354-8518
 43 M 70402 68 IM
 SUTTON, RICHARD J, 1706 W TENTH,66604
 235-2311
 38 M 4706 67 DRS
 SWOGER JR, GLENN, MENNINGER FD,66601
 273-7500
 35 M 3806 60 P
 TAPPEN, DANIEL L, 2849 MACVICAR,66611
 -
 16 M 1902 41 DD
 TARGOWNIK, KARL K, 1218 W TENTH,66604
 232-1644
 15 M 40710 49 P
 TARNOWER, WILLIAM, MENNINGER CLINIC,66601
 273-7500
 21 M 4802 48 P
 TEMPERD, STEPHEN J, 310 MED ARTS BLDG,66604
 234-3451
 42 M 1606 67 R
 THOMS, NORMAN W, 40 MED ARTS BLDG,66604
 233-1710
 34 M 2501 59 TS
 THURSTON, DAVID E, 200 CONTINENTAL BLDG,66606
 233-7491
 29 M 1902 55 DRS
 TIETZE, DENNIS D, 634 MULVANE STE 402,66606
 235-9545
 50 M 1902 78 FP
 TOTH, JOHN R, 2115 W TENTH,66604
 232-3330
 49 M 1902 71 FP
 TOZER, RICHARD C, 1447 DAKLEY,66604
 -
 19 M 4102 45 DD
 TRAVIS, JOHN W, S-F CAP REG RADIOOTHER CTR,66606
 295-8008
 29 M 1606 55 R

TREES, CLYDE B. 3700 HUNTDON.66604

09	M	2401	33	DD
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 TREGER, NEWMAN V. 1704 W 10TH.66604
 354-8761

16	M	1902	40	IM
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 TUCKER, VIRGINIA L. BUREAU DF MAT & CHILD HLT.66620
 862-9360

30	F	1902	57	PD
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 TWEMLDW, STUART W. 707 QUINCY SUITE 208.66603
 233-1607

41	M	67101	73	P
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 UHR, NATHANIEL. MENNINGER FOUNDATION.66601
 273-7500

00	M	3519	21	IM
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 VAN SICKLE, GREGGORY J. 634 MULVANE.66606
 233-8508

49	M	1606	75	PD
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 VANDE GARDE, LARRY D. 800 LINDOLN.66606
 233-5101

41	M	1803	66	DBG
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 VDGL, STANLEY J. 901 GARFIELD.66606
 354-9591

44	M	2802	70	DNC
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 WADE III, WILLIAM E. D.D., 1115 W TENTH SUITE A.66604
 233-8268

53	M	3979	80	FP
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 WALLACE, LED F. 5500 W 24TH.66614

17	M	1902	41	DD
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 WALLS, WILLIAM J. 310 MED ARTS BLDG.66604
 354-6171

39	M	2834	66	DR
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 WALZ, RODYCE C. 1710 W 10TH SUITE 205.66604
 234-2676

27	M	15407	60	P
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 WANLESS, KIRK M. 1424 W EIGHTH.66606
 232-8188

44	M	2803	74	DTD
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 WARD, HOWARD N. 901 GARFIELD.66606
 354-9591

37	M	1606	62	HEM
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 WARE, LUCILE M. MENNINGER FOUNDATION.66601
 273-7500

29	F	3501	53	P
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 WARRICK, DAVID ALAN. 600 MADISON.66607
 354-5275

49	M	3843	76	IM
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 WATERS, DALE A. 634 SW MULVANE SUITE 203.66606
 233-1690

41	M	5605	67	CDS
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 WEAVER, WALTER D. 900 WASHBURN ST.66606
 233-3636

41	M	1902	69	DPH
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 WEBER, DARRELL J. 1710 W 10TH.66604
 233-2305

16	M	1902	44	FP
----	---	------	----	----

 WEST, CHARLES M. 901 GARFIELD.66606
 354-9591

47	M	3841	73	IM
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 WILCOX, DONALD E. 122 DANBURY LANE.66606
 296-3782

24	M	1902	55	PH
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 WILSON, MARVIN H. 1516 SW SIXTH.66606
 233-1747

38	M	1003	64	GS
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 WINER, RICHARD S. 5813 SW 22ND TERR #4.66614
 273-7500

55	M	1902	80	P
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 WDNG, NORMUND. MENNINGER FD.66601
 273-7500

34	M	502	59	P
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 WDDO, EDWARD RUSSELL. 901 GARFIELD.66606
 354-9591

49	M	1902	71	IM
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 WDDOS, ROBERT P. 901 GARFIELD.66606
 357-6171

14	M	6701	40	N
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 YEH, ROBERT M. 2315 W 34TH.66611
 235-3451

47	M	24405	73	ANES
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 YORKE JR, CRAIG H. 901 GARFIELD.66606
 357-6171

48	M	2401	74	NS
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 YOUNG, PAUL E. TOPEKA MEDICAL CENTER.66604
 233-4927

42	M	2407	75	JPH
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 YOUNG, THEODORE E. 107 MED ARTS BLDG WEST.66604
 232-0576

22	M	2307	46	PD
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 ZACHARIAS, DAVID LLOYD. 1500 W TENTH.66606
 354-6870

26	M	1902	53	PATH
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 ZIMMERMAN, WILLIAM H. CONTINENTAL BLDG.66606
 232-4377

20	M	3006	52	GS
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TOWANDA — 316

(Sedgwick County Society)

 NYBERG, FREDRIK F. ROUTE 1.67144

22	M	2101	46	DD
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TRIBUNE—316

(Southwest Kansas Society)

 WERNER, WILLARD F. .67879
 376-4251

24	M	1902	52	FP
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TROY—913

(Northeast Kansas Society)

 MASTERSON, MELVIN LERDY. 210 S MAIN.66087
 985-2211

23	M	4901	48	R
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ULYSSES—316

(Southwest Kansas Society)

 BREWER, MARSHALL A. PD 8DX 687.67880
 356-1261

19	M	1902	46	FP
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 RAMCHANDANI, A P. 301 E GRANT.67880
 356-2432

39	M	49530	74	FP
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 TILLOTSON, DON R. 8DX 687.67880
 356-1261

32	M	1902	65	FP
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VALLEY CENTER—316

(Sedgwick County Society)

 DANIELS, ROBERT M. BDX 12B.67147
 262-6262

24	M	1902	54	FP
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 WILSON, ROBERT L. RR 1.67147
 685-2563

30	M	1902	57	EM
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WAKEENEY—913

(Central Kansas Society)

 BERNER, NEAL E. 418 B RUSSELL.67672
 743-2124

44	M	1902	72	FP
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 HAMILTON, JAMES J. MEDICAL CENTER.67672
 743-2124

30	M	1902	55	FP
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WAMEGO—913

(Pottawatomie County Society)

 BORGENDALE, LLEWELLYN V. 507 ELM.66547
 456-2291

29	M	1902	60	FP
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 BRADEN, BILL L. PD 8DX 7.66547
 456-2291

31	M	1902	60	FP
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 CLARK, LAURENCE A. 507 ELM.66547
 456-2291

12	M	1902	42	FP
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 WELCH, MAURA S. 8DX 149.66547
 456-9202

50	F	1902	75	OBG
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 WIGGLESWORTH, ANNE. HWY 24 PD BDX 149.66547
 456-9202

40	F	1902	75	OBG
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WASHINGTON—913
(Northeast Kansas Society)

BITZER, DONALD A. 115 W 3RD, 66968
-
03 M 3005 26 00
HODGSON, DAVID K. 107 E THIR, 66945
325-2259
49 M 1902 78 FP

WATHENA—913
(Northeast Kansas Society)

PETERSON JR, EVAN A. 324 ST JOSEPH ST, 66090
989-3122
24 M 1803 55 FP

WELLINGTON—316
(Tri-County Society)

ANDERSON, LARRY R. 1323 NORTH A, 67152
326-3301
43 M 1902 73 FP
COLE, WARD M. 110 N JEFFERSON, 67152
326-7221
08 M 1902 36 FP
MALOZZA JR, FAUSTINO M. 1323 NORTH A, 67152
326-8171
M 74801 64 GS
PEDRAZA, HERNANDO, 33 CRESTWAY, 67152
326-8070
28 M 26404 56 R
WEIGANO, JOEL T. 1323 NORTH A, 67152
326-3301
43 M 1902 70 FP

WESTMORELAND—913
(Pottawatomie County Society)

DECHAIRD, THOMAS, DECHAIRD HOSP, 66549
457-3311
13 M 1902 36 FP

WICHITA—316
(Sedgwick County Society)

ABBAS, OILAWER M. 1515 S CLIFTON, 67218
685-1111
45 M 70402 71 M
ACEVEDO, ALFREDO, 959 N EMPORIA SUITE 205, 67214
255-4701
40 M 73701 59 COTS
AGUSTIN, CONRADO M. 1035 N EMPORIA STE 165, 67214
267-3389
38 M 74807 62 OBG
AHLSTRAND, RICHARD A. 3243 E MURDOCK SUITE 104, 67208
685-2711
41 M 3005 67 R
ALDOODY, NEIL, 3243 E MURDOCK STE 400, 67208
686-7351
46 M 54914 75 P
ALEXANDER, ELIZABETH, 3243 E MURDOCK SUITE 300, 67208
585-8231
46 F 1902 77 FP
ALFONSO, MANJEL, 3244 E DOUGLAS, 67208
689-9445
37 M 84710 66 ANES
ALLEN, PHILLIP M. WESLEY MEDICAL CENTER, 67214
688-2838
27 M 2401 54 PATH
ALMONTE, PRISCILLA C. 303 S HILLSIDE, 67211
584-7251
44 F 74801 67 ANES
ALMONTE, RODOLFO O. 1515 S CLIFTON SUITE 480, 67218
686-3791
39 M 74801 64 OBG
AMMAR, ALEX D. 818 N EMPORIA SUITE 200, 67214
263-0296
51 M 5101 76 GPVS
ANDERSON, EUGENE G. SUTTON PLACE, 67202
265-8619
19 M 1902 44 OBG

ARGOSINO, RODOLFO, 1148 S HILLSIDE, 67211
683-6506
40 M 74801 63 GS
ARNOLD, KATH-EEV J. 3701 EDGE MONT, 67208
-
55 F 1902 84
ARTZ, TYRONE D. 1125 N TOPEKA, 67214
267-0362
41 M 1803 67 ORS
ASHMORE, ARTHUR L. 811 N MISSION, 67206
-
35 M 1902 32 TS
AUNINS, JOHN, 4853 HEMLOCK, 67216
524-6805
28 M 4706 56 FP
BACKES, DAVID J. 851 N HILLSIDE, 67214
635-8321
48 M 1720 77 U
BAILEY, DONALD C. 3243 E MURDOCK, 67208
685-1491
37 M 3901 65 ORS
BAMMEL, BRUCE, 3244 E DOUGLAS, 67208
689-9234
52 M 2507 78 JBG
BARBA, ESTRELA G. 8309 BROOKHOLLOW LANE, 67206
264-2301
41 F 74802 66 CHP
BARBA JR, ANTONIO P. 1035 N EMPORIA STE 280, 67214
264-2301
34 M 74807 62 OBG
BARKER, BENJAMIN W. 8015 WILLOWBROOK, 67207
-
18 M 1902 51 00
BARKER, MONTY R. 848 S CHAUTAQUA, 67211
-
57 M 1902 85
BARKER, PATSY, 818 N EMPORIA SUITE 303, 67214
265-3774
49 F 64914 75 PD
BARNES, JOE L. 1805 GRIFFITH, 67207
689-8070
54 M 1902 82 FP
BARNETT, ARNOLD M. 5111 E 21ST, 67208
685-2561
32 M 83601 54 M
BARTAL, ELY, 818 N EMPORIA SUITE 306, 67214
262-7598
45 M 39607 70 ORS
BARTLETT, WAYNE C. 13 HAMPTON RD, 67207
689-9124
07 M 1601 31 GS
BASINGER, BRADLEY B. 350 N ERIE, 67214
-
53 M 1902
BASS II, ORAL E. 851 N HILLSIDE, 67214
685-1371
40 M 2803 71 U
BASSELL, G M. 3333 E CENTRAL SUITE 602, 67208
685-4389
45 M 14303 73 ANES
BATES, MICHAEL D. 2703 EAST CENTRAL, 67214
685-1277
48 M 3005 74 OBG
BATTISTE, CYNTHIA ELAINE, 3333 E CENTRAL SUITE 416, 67208
691-2021
47 F 1606 73 PD
BAUMAN, M LEON, 6042 E 13TH ST, 67208
-
01 M 1902 44 00
BAUMANN, PAUL A. 3333 E CENTRAL, 67208
685-1291
32 M 5605 57 R
BEBAK, DONALD M. 2322 E CENTRAL, 67214
263-6186
32 M 3515 58 ANES
BECK, CHARLES W. 1148 S HILLSIDE SUITE 10, 67211
685-8262
46 M 301 72 1M
BECKER, KARL E. 818 N EMPORIA, 67214
268-6151
43 M 2307 69 ANES
BETHEL, CHANDLER S. 5107 E 21ST ST, 67208
682-6559
34 M 1902 59 1M
BHARGAVA, BAIKUNTH N. 3243 E MURDOCK SUITE 102, 67208
682-4523
37 M 49530 63 U
BIBERSTEIN, GREG A. 1739 LEXINGTON, 67218
-
56 M 1902 84
BIERMANN, HENRY J. 425 E MURDOCK, 67214
265-6287
27 M 3006 52 GS

BIERMANN, WILLIAM J, 1435 LEONETT, 67203

04 M 3006 29 00
 BINGAMAN, ROBERT W, 7111 E 21ST, 67206
 682-1053

47 M 3901 72 GS
 BINYON, KERVIE W, 4618 E CENTRAL, 67208
 684-2819

24 M 1902 56 FP
 BLATT, GEOFFREY L, 1329 FARMSTEAD, 67208

56 M 1902 84
 BLAYLOCK, HOYT C, 835 N HILLSIDE, 67214
 685-4395

21 M 1902 45 D
 BLOOM, ROONEY LAMONT, 2129 GEORGE WASHINGTON DR, 67218

54 M 1902 79 IM
 BLOXHAM, THOMAS J, 3244 E DOUGLAS, 67208
 689-9215

50 M 1803 75 PUD
 BOCK, PETER A, 1542 FAIRVIEW, 67203

57 M 1902 84
 BOEHM, DOUGLAS K, 3243 E MURDOCK SUITE 500, 67208
 684-0251

52 M 1902 77 IM
 BOEHM, MINOY M, 3333 E CENTRAL SUITE 408, 67208
 682-0411

52 F 1902 78 PD
 BOYO, ROGER C, 3243 E MURDOCK, 67208
 684-0251

40 M 5606 67 CO
 BOYD, Z REX, 120 S MAIZE RD #12, 67209
 268-5000

26 M 3005 52 FP
 BOYLE, HUGH H, 424 N WOODLAWN, 67208
 696-2193

33 M 3806 60 PATH
 BRAKE, DAVID, 3243 E MURDOCK SUITE 104, 67208
 685-2711

43 M 702 68 R
 BRAUN, KENNETH, 1431 S BLUFFVIEW, 67218
 683-4688

47 M 3519 72 OPH
 BRAUN, THOMAS G, 3243 E MURDOCK #601, 67208
 685-2377

35 M 6001 61 N
 BRAUN III, WILLIAM T, 3243 E MURDOCK SUITE 104, 67208
 585-2711

37 M 2802 61 R
 BRECKBILL, DAVID L, 3333 EAST CENTRAL #214, 67208
 685-1291

39 M 1902 64 R
 BRENNEIS, ANN T, 400 N CENTRAL APT 3115, 67203

60 F 1902 86
 BRINTON, E HOLMES, 3244 E DOUGLAS, 67208
 689-9124

46 M 2101 70 GS
 BRINTON, EDWARD S, 329 NORTH TERRACE DR, 67208

15 M 1611 41 00
 BRITO, RAUL E, 3243 E MURDOCK, 67208
 682-4523

32 M 31901 59 U
 BROSIUS, FRANK C, 3243 E MURDOCK, 67208
 684-0251

25 M 1902 49 IM
 BROWN, DAVID J, 425 EAST MURDOCK, 67214
 265-6287

45 M 1902 71 GS
 BROWN, MICHAEL P, 345 N HILLSIDE, 67214
 683-6766

51 M 3007 77 OBG
 BROWN, ROBERT L, 5025 E KELLOGG, 67218
 682-1534

21 M 1902 49 FP
 BROWN, RONALD C, 3243 E MURDOCK, 67208
 695-8231

47 M 2803 73 FP
 BROWN, ROYALD L, 1128 S CLIFTON, 67218
 684-7251

45 M 3901 71 ANES
 BROWN, VAL J, 1802 N HYDRAULIC, 67214
 265-1461

24 M 1003 47 FP
 BROWN JR, VAL J, 1802 N HYDRAULIC, 67214
 265-1461

53 M 1902 79 IM
 BROWNING, WILLIAM H, 7077 E CENTRAL #17, 67206

16 M 1902 43 00

BUBECK, RALPH W, 3244 E DOUGLAS, 67208
 689-9396

36 M 1803 62 IM
 BUCK JR, BEN H, 1515 S CLIFTON SUITE 480, 67218
 684-1048

17 M 2834 43 TS
 BULGER, ROSE M, 916 N DELLROSE, 67208

48 F 1902 84
 BULLER, DAVID L, 9000 E LINCOLN #2203, 67207

58 M 1902 85
 BURGARD, PETER A, 1907 LEXINGTON, 67218

54 M 1902 82 FP
 BURNEY, WILLIAM W, 1755 N MADISON, 67214
 264-8311

17 M 1902 52 FP
 BURNEY II, WILLIAM W, 1755 N MADISON, 67214
 264-8311

50 M 4707 76 IM
 BURPEE, JAMES F, 851 N HILLSIDE, 67214
 585-1371

39 M 5605 66 U
 BURT, RONALD J, 1628 52ND NW, 67204

47 M 1902 84
 BUTH, DENNIS K, 2916 EAST CENTRAL, 67214
 684-5243

45 M 1902 72 IM
 BUTIN, J WALKER, 3244 E DOUGLAS, 67208
 589-9477

23 M 1902 47 IM
 BUTLER, DORIS C, 1148 S HILLSIDE, 67211
 684-2329

48 F 1902 75 FP
 BYRNE, JAMES PERRY, 818 N EMPORIA SUITE 200, 67214
 263-0296

42 M 2101 68 TS
 CALIENDO JR, DANIEL J, WESLEY MED CENTER, 67214
 685-2563

41 M 1902 67 EM
 CANNON, DONALD C, 5400 E 21ST APT 108, 67208

34 M 1602 60 IM
 CANNON, MICHAEL W, 1035 N EMPORIA SUITE 265, 67214
 588-6029

50 M 1902 75 ON
 CAPPER, STANLEY L, 3244 E DOUGLAS, 67208
 689-9206

37 M 1803 67 D
 CARSON, TERRY S, 550 N HILLSIDE, 67214
 688-2820

50 M 3006 77 PATH
 CARSON, BERTRAM H, D.O., ST FRANCIS HOSP, 67214
 268-5050

26 M 2878 64 EM
 CARTER, MACK A, 655 N WOODLAWN, 67208
 684-5158

18 M 1902 50 OPH
 CASTELLANI, SAM, UKSM WICHITA PSY DEPT, 67214
 261-2647

41 M 2507 67 P
 CAUBE, WILBUR G, 1148 S HILLSIDE SUITE 102, 67211
 693-1681

12 M 2834 39 GS
 CAUGHLIN, GERALD MICHAEL, 818 N EMPORIA SUITE 307, 67214
 268-6189

55 M 4812 80 ANES
 CAWLEY, LEO P, WESLEY MED CENTER, 67214
 685-2151

22 M 3901 52 PATH
 CHAVEY, ERNIE J, ST JOSEPH MED CENTER, 67218
 685-1111

27 M 1902 56 FP
 CHANG, FREDERIC C, 818 N EMPORIA SUITE 200, 67214
 263-0296

35 M 2401 59 GS
 CHAPMAN, JAMES H, PO BOX 2579, 67201
 265-1684

27 M 4706 63 R
 CHARD, FREDERICK H, 3244 E DOUGLAS, 67208
 689-9129

15 M 5605 39 D
 CHI, IL-SUNG, 3333 E CENTRAL STE 602, 67208
 685-4389

41 M 58302 67 ANES

CHO, SECHIN, UKSM - WICHITA, 67214
 261-2631

47 M 58302 71 PD

CHOPRA, RAMAN, 3333 E CENTRAL #201, 67208
 685-5271

52 M 49536 76 PD

CHRISTMAN JR, CARL, 550 N LORRAINE, 67214
685-0559

48 M 4802 74 OBG
CLARK, COURTNEY, 303 S HILLSIDE, 67211
684-7251

30 M 1902 56 ANES
CLIFTON, H DAVID, 3600 E HARRY, 67218
685-1111

41 M 401 65 R
CLINE, BYRON W, 550 N LORRAINE, 67214
585-0559

51 M 4802 77 OBG
COHEN, JUSTIN THOMAS, 655 N WOODLAWN, 67208
684-5158

47 M 2803 74 OPH
COHMIA, JERRY B, 818 N EMPORIA SUITE 310, 67214
263-5891

43 M 1902 70 IM
COLEMAN, THOMAS J, 959 N EMPORIA, 67214
265-0749

18 M 3545 51 IM
COLLIER, HAROLD W, 1515 S CLIFTON SUITE 260, 67218
683-5008

45 M 1902 71 ANES
CONDANNON, CRAIG A, 202 N ROCK RD APT 1307, 67206
-

58 M 1902 84
CONCEPCION JR, EUGENIO S, 1515 S CLIFTON SUITE 480, 67218
684-1048

39 M 74802 64 CD
CONRADY, PETER A, 818 N EMPORIA #101, 67214
686-7327

42 M 515 69 ANES
COOK, DONALD RAY, 315 N HILLSIDE, 67214
686-3391

42 M 2012 71 FP
CDDK, G EDWARD, 5T JOSEPH HOSPITAL, 67218
685-1111

42 M 401 67 R
COOPER, M KEV, 818 N EMPORIA STE 307, 67214
268-6189

54 M 1902 79 ANES
CORDRY, VINCEL R, D.O., 1512 MAYBELLE, 67212
-

47 M 2878 75 P
CORDONADD, EDWARD H, 1945 N ROCK RD APT 2219, 67206
-

51 M 74808 76 P
COSSMAN, F PRICE, 851 N HILLSIDE, 67214
685-1371

28 M 1902 57 U
COWLES, GORDON T, 3333 E CENTRAL, 67208
683-2661

32 M 1902 58 OBG
CRANE, DAVID D, 929 N ST FRANCIS, 67214
252-6211

34 M 2501 60 PATH
CRONIN, DONALD J, 3244 E DOUGLAS, 67208
689-9227

16 M 2604 40 ENT
CROW, ERNEST W, 3243 E MURDOCK, 67208
684-0252

20 M 1902 44 CD
CROWLEY, EDWARD X, 345 N HILLSIDE, 67214
582-4519

14 M 1643 39 GYV
CUMMINGS, RICHARD J, 427 N HILLSIDE, 67214
686-6608

32 M 1902 57 OTD
DAKHIL, SHAKER R, 818 N EMPORIA SUITE 403, 67214
262-4467

50 M 60501 75 IM
DANBY, JOHN H, 905 N EMPORIA, 67214
265-5996

29 M 35205 56 FP
DANSILL, DAVID J, 350 N ERIE, 67214
-

M 1902 85
DARRAM, CAROL J N, 3333 E CENTRAL SUITE 613, 67208
685-9289

49 F 1902 74 R
DAVIDSON, HARRY T, 556 N BROADVIEW, 67208
-

87 M 3802 11 OO
DAVIS, PAUL H, 7111 E 21ST, 67206
684-2851

47 M 3901 72 FP
DAVIS, RONALD B, 1148 S HILLSIDE, 67211
685-2153

46 M 1902 72 FP
DAY, HOWARD, 818 N EMPORIA SUITE 310, 67214
263-5891

48 M 1902 74 NEP

DE BAKKER, JAN B, 1035 N EMPORIA, 67214
263-4903

25 M 5104 59 GS
DE HART, ARTHUR DONIVA, 2703 E CENTRAL, 67214
685-1277

50 M 4804 77 OBG
DEGNER, JAMES C, 4901 ARLENE, 67220
-

57 M 1902 84
DEJOY, DAVID C, 550 WEST SHORE DR, 67209
268-5424

33 M 2501 59 PATH
DEMOSS, ELEANOR P, 3333 E CENTRAL SUITE 407, 67208
682-5591

42 F 74802 66 PD
DIACON, JAMES L, 2029 N WOODLAWN #710, 67208
326-2111

24 M 3901 52 FP
DIAZ, DOLores M, 2579 GREENLEAF CT, 67226
686-7327

51 F 64902 75 ANES
DIAZ, SALVADOR F, 3333 E CENTRAL STE 602, 67208
685-4389

45 M 64902 73 ANES
DIRKSEN, HANS C, 550 N HILLSIDE, 67214
689-2360

47 M 6001 71 NEO
DOEBLIN, P LAURENCE, 3333 E CENTRAL SUITE 214, 67208
585-1291

40 M 1002 73 R
DOLAN JR, PHILIP JARVIS, 3244 E DOUGLAS, 67208
689-9477

47 M 2105 73 GE
DONATELLE, EDWARD P, UKSM WICHITA, 67214
268-8221

22 M 2604 50 FP
DOMLEY, JAMES L, 3101 E NINTH, 67208
684-0201

46 M 1902 72 P
DONNELL, JAMES F, 449 N YALE, 67208
-

54 M 1902 84
DONNELL, JAMES M, 2320 E CENTRAL, 67214
264-8010

28 M 1902 55 FP
DOORNBOOS, J FRED, 929 N ST FRANCIS, 67214
262-6211

28 M 1902 57 R
DOUGHERTY JR, THOMAS M P, 343 N HOLYOKE, 67214
-

56 M 1902
DOUTHIT, DOUGLAS DAVID, 550 N LORRAINE, 67214
685-0559

53 M 4802 79 OBG
DRAKE, RALPH L, 4422 E 3RD, 67208
-

99 M 4102 26 DO
DRAZEK, GEDRGE, 3244 E DOUGLAS, 67208
689-9111

50 M 3506 76 OPH
DREVETS, CURTIS C, 3244 E DOUGLAS, 67208
689-9178

30 M 1902 56 IM
DUICK, GREGORY, 1035 NORTH EMPORIA #130, 67214
263-3271

46 M 1643 72 CD
DUNSHIE, CHERYL A, UKSM WICHITA, 67214
268-8378

54 F 1902 79 IM
DURANO, ANTONIO C, 959 N EMPORIA, 67214
263-7893

29 M 74807 56 U
DYER, VERNON E, 3244 EAST DOUGLAS, 67208
262-6202

36 M 301 72 OBG
ECK, MARCI J, 1945 N ROCK RD SUITE 2208, 67206
-

59 F 1902 86
ECKERT, WILLIAM G, MILTON HELPERN CTR BOX 95, 67206
689-3707

26 M 3519 52 PATH
EDWARDS, MANIS C, 3333 E CENTRAL, 67208
583-2661

33 M 3005 58 OBG
EGBERT, ANNE MARSH, UKSM WICHITA, 67214
261-2650

54 F 3840 79 IM
EGELHOF, RICHARD H, 1035 N EMPORIA SUITE 290, 67214
263-1299

45 M 1902 73 FP
ELANGOVAN, SUDHA, 202 N ROCKROAD SUITE 611, 67206
-

45 F 1902 87

ELLIS, HARVEY D. 5611 E CENTRAL.67206

683-1022

24 M 1902 55 GS

ELLIS, JOSEPH G. 3244 E DOUGLAS.67208

689-9132

25 M S11 62 1M

ELNEN, WALTER T. 460 N TERR DR.67208

682-5671

03 M 1643 32 GS

ENOCH, ROLLAND. 315 N HILLSIDE.67214

651-0423

49 M 64914 76 FP

ERKEN, RONALD V. WICHITA PSYCHIATRIC CTR.67208

684-0201

29 M 2834 56 P

ERNST, R L. 803 FRANKLIN.67203

-

56 M 3005 82 GS

ERNST, TARI MAE. 1010 N KANSAS.67214

261-2622

56 F 3005 81 FP

ESTEP, THOMAS H. 818 N EMPORIA SUITE 200.67214

263-0296

51 M 6002 75 CO

EVANS, FARRIS D. 521 RUTLAND RD.67206

-

05 M 1902 32 FP

EVANS, GRANT E. 2703 E CENTRAL.67214

682-6556

21 M 4901 46 FP

EVANS, JOHN F. 5826 POLD.67208

688-2360

42 M 2803 70 MFM

EVANS, RICHARD W. UKSM WICHITA.67214

268-8378

51 M S107 76 1M

EVANS, ROGER WILLIAMS. 933 N TOPEKA.67214

263-5889

39 M 1902 64 CO

EYSTER, ROBERT L. 3243 E MURDOCK.67208

685-1491

47 M 3901 73 ORS

FARMA, GEORGE J. 818 N EMPORIA SUITE 200.67214

263-0296

27 M 2101 57 GS

FARMA, S JIM. 818 N EMPORIA SUITE 200.67214

263-0296

31 M 1001 57 TS

FARLEY, JAMES A. ST JOSEPH MEDICAL CENTER.67218

685-1111

50 M 1902 78 PATH

FENDER JR. THOMAS H. 3432 EVERETT.67217

267-8439

25 M 4812 54 NP

FERRELL, DONALD P. ST JOSEPH MEDICAL CTR.67218

685-2371

36 M 3901 63 EM

FERRER, ROGER W. 3244 E DOUGLAS.67208

689-9445

48 M 3806 80 ANES

FERRIS, BRUCE G. 825 N HILLSIDE.67214

688-7500

43 M 1902 69 PS

FEUILLE JR. EDMOND G. 212 N HILLSIDE.67214

692-4572

50 M 4802 75 JBG

FIELOS, STEPHEN, D.O.. 7200 W 13TH.67212

722-4258

42 M 2878 72 FP

FINLEY, DENNIS R. 1035 N EMPORIA.67214

262-7429

35 M 1606 62 ORS

FISHER, JAMES B. 141 S OLD MANOR.67218

-

09 M 1902 36 OO

FISHER, RAY F. 3243 E MURDOCK SUITE 500.67208

684-0251

49 M 1902 74 1M

FITZGERALD, EDWARD J. 3600 E HARRY.67218

685-1111

22 M 3006 50 R

FITZIG, SANFORD. 3244 E DOUGLAS.67208

689-9344

46 M 4102 72 U

FLEMING, FORNEY W. 3243 E MURDOCK #200.67208

685-1491

43 M 4802 69 ORS

FLOWERS JR, CLELL B. 855 N HILLSIDE.67214

685-1381

22 M 1902 55 FP

FORD, CHARLES R. 232 S MAIZE RD.67209

722-0568

38 M 1902 63 OPH

FOWLER, ROBERT J. 3244 E DOUGLAS.67208

689-9236

37 M 2802 63 1M

FRANCIS, NORTON L. 55 VIA ROMA.67230

-

10 M 3005 35 ENT

FRANCISCO, DAN A. 3243 MURDOCK SUITE 500.67208

684-0251

40 M 1803 75 CO

FRANCISCO, LINDA L. 818 N EMPORIA SUITE 310.67214

263-5891

47 F 1803 74 NEP

FRANTZ, SHIRLEY J. 10300 W MAPLE.67209

722-6260

55 F 1611 80 FP

FRENCH, JAMES E. 1515 S CLIFTON SUITE 260.67218

684-5237

53 M 3005 78 GS

FRENCH, JEROME E. MIO-KANSAS ENT ASSOC.67211

684-2838

44 M 1103 71 OTO

FRITZEMEIER, WILLIAM H. 835 N HILLSIDE.67214

685-4395

14 M 1902 41 O

FROMER, JOEL. 2627 E CENTRAL.67214

654-2501

46 M 16501 75 A

FROMM, ARTHUR H. 315 N HILLSIDE.67214

685-2281

37 M 1902 63 FP

FUGATE, CARL L. 2248 PATTIE.67211

-

57 M 1902 84

FULTON, JOHN K. 3333 E CENTRAL SUITE 801.67208

686-0732

18 M 5605 43 PUD

GALICHIA, JOSEPH P. PO BOX 594.67201

263-3271

42 M 1902 69 CO

GALVAN, ALONSO. 3243 E MURDOCK.67208

684-0251

39 M 64906 64 1M

GATSCHET, TIMOTHY P. 314 COMMERCIAL PO BOX 295.67135

-

50 M 1902 85

GENILO, AMANCIO C. 120 LONGFORD CT.67206

685-2371

37 M 74801 61 EM

GENILO, CELESTE A. 3244 EAST DOUGLAS.67208

689-9445

39 F 74801 62 ANES

GEORGE, EARL F. 5107 E 21ST.67208

265-6991

35 M 1902 65 FP

GEORGE, M OON. #1CHITA PSYCHIATRIC CL.67208

684-0201

31 M 1001 56 P

GERBER, ALLEN D. 7111 E 21ST.67206

692-1053

48 M 1902 71 GS

GILMARTIN, RICHARD C. 3243 E MURDOCK #601.67208

685-2377

32 M 4112 58 POH

GIVNER, DAVID. 2627 E CENTRAL.67214

684-0501

03 M 2301 29 1M

GOERING, RANDALL V. 401 S LORRAINE.67211

-

58 M 1902 84

GOHIL, MAHENDRA N. 1823 RUTLAND.67206

265-1684

28 M 40930 66 OR

GOLOSBERG, HERBERT R. 400 WOODLAWN SUITE 101.67208

685-4215

33 M 3508 59 PO

GONZALEZ, HIRAM. 3429 E DOUGLAS.67218

681-1348

20 M 64901 52 P

GOODPASTURE, HEWITT C. 818 N EMPORIA SUITE 305.67214

264-3505

43 M 1902 69 1M

GORDON, JAMES R. 3244 E DOUGLAS.67208

689-9260

53 M 1611 78 1M

GOYLE, KRISHAN K. 1144 N ST FRANCIS.67214

267-0159

34 M 49529 63 CO

GOYLE, VIMAL. 1150 N ST FRANCIS.67214

267-9906

41 F 49529 65 DBG

GRAUEL, CHARLES W. 8310 CHALET.67207

262-1127

44 M 1902 70 ANES

GRAVES, JACK W. 3244 E DOUGLAS, 67208

17 M 1902 42 DO

GRAY, C LUCIEN, 4821 E CENTRAL, 67208

684-5171

21 M 1902 45 ENT

GRAY, H TOM, 3610 E ENGLISH 1028, 67218

19 M 401 44 DO

GREER, JAMES A. 3244 EAST DOUGLAS, 67208

659-9227

43 M 1611 69 OTO

GRENE, ROBERT BRUCE, 655 N WOODLAWN, 67208

684-5158

53 M 1902 78 OPH

GRIBBLE, ROBERT N. 1035 N EMPORIA #285, 67214

264-2401

43 M 1902 69 ?

GRILLLOT, FLOYD B. 1515 S CLIFTON SUITE 150, 67218

684-0243

18 M 1902 51 FP

GROHS, HEINZ K. 550 NORTH HILLSIDE, 67214

685-2151

42 M 15407 66 PATH

GRUND, FRANK M. 3244 EAST DOUGLAS, 67208

689-9420

47 M 1803 72 IM

GRUSHNYS, ARNOLD, 3244 E DOUGLAS, 67208

689-9445

19 M 40721 59 ANES

GSE., GEORGE F. 32 MISSION RD, 67206

07 M 1601 33 DO

GUTHRIE, RICHARD A. KS REG DIABETES CENTER, 67214

268-8228

35 M 2803 60 PO

GWINN, DOUGLAS R. 514 N HOLYOKE #204, 67208

53 M 2803 80 FP

HABASHY, SHAWKY N F. 905 N EMPORIA, 67214

268-5996

43 M 33004 65 OBG

HAGAN, C THOMAS, 959 N EMPORIA, 67214

265-0789

16 M 3006 42 IM

HAGAN, FRANCIS J. PO BOX 1837, 67201

262-1057

13 M 3006 39 FP

HAGAN, ROBERT C. 3244 E DOUGLAS, 67208

689-9306

52 M 1902 77 GE

HALE, RALPH, 847 S HILLSIDE, 67211

684-0295

18 M 1902 46 A

HALL, J ROGER, 1515 S CLIFTON SUITE 310, 67218

685-5227

42 M 4802 68 OPH

HALPIN, EDWARD O. 1148 S HILLSIDE, 67211

685-9229

33 M 1902 62 OBG

HANSON, ROBERT L. 6202 ONEIDA, 67208

59 M 1902 86

HARMS, EDWIN M. 5623 POLO DR, 67208

06 M 3901 34 DO

HARRIS, FRANK H. 1035 N EMPORIA, 67214

09 M 1001 39 NP

HARRISON, PAUL BARRY, 3243 EAST MURDOCK, 67208

685-6222

49 M 1902 74 GS

HARSTINE, LILLIAN R. 3243 E MURDOCK SUITE 404, 67208

682-6585

51 F 1902 76 IM

HART, DILLIS L. 1515 S CLIFTON SUITE 300, 67218

698-0135

36 M 3901 64 GS

HART, JOHN J. 3243 E MURDOCK SUITE 303, 67214

689-5775

53 M 74808 60 GP

HARTLEY, JAMES M. 3243 E MURDOCK SUITE 300, 67208

685-8231

45 M 2604 71 FP

HARTWELL, RICK L. 3243 E MURDOCK SUITE 303, 67204

83 M 1902 82 FP

HARVEY, ROSEMARY B. 635 N MAIN, 67203

268-8025

24 F 1902 49 ADM

HASAN, SANJIDA. 6602 E HARRY RD APT 904, 67207

41 F P

HASSAN, RIZWAN U. 1515 S CLIFTON STE 360, 67218

686-2831

47 M 70305 70 N

HATESOHL, STANLEY M. 900 N WACO #109, 67203

264-9863

57 M 1902 84

HATTRUP, RICHARD J. 610 N TYLER, 67212

722-8837

31 M 3006 57 FP

HAWLEY, RAYMOND G. 1451 N WOODLAWN, 67208

685-1111

39 M 1902 65 PATH

HAYES, WILLIAM L. 3243 E MURDOCK, 67208

684-0251

28 M 1902 53 CO

HAYNES, JEBORAH G. 1131 S CLIFTON, 67218

685-4354

54 F 1902 79 FP

HAYS, THOMAS H. 7111 E 21ST, 67206

684-2851

49 M 1902 75 FP

HEADRICK, DAVID E. 2022 S RIDGEWOOD, 67218

56 M 1902 83 ANES

HENNING, CHARLES E. 320 N HILLSIDE, 67214

682-3221

37 M 1902 63 ORS

HERED, JOHN. 959 N EMPORIA SUITE 403, 67214

262-3613

41 M 2802 67 N

HERSHBERGER, GROVER, D.O., 1245 N WEST ST, 67203

945-6910

47 M 2878 79 GP

HERSHORN, SIMON E. 3333 E CENTRAL, 67208

685-1291

22 M 1902 46 R

HIEBERT, ABRAHAM E. 1530 W 13, 67203

94 M 2802 25 DO

HILL, RILEY M. 929 N ST FRANCIS, 67214

55 M 3901 82 IM

HINSHAW, ALFRED H. 6110 ONEIDA, 67208

07 M 1902 33 DO

HINSHAW, CHARLES T. 256 N BLECKLEY DR, 67208

99 M 4705 26 DO

HINSHAW JR, CHARLES T. 3715 E DOUGLAS, 67218

684-7784

32 M 1902 58 PATH

HINZE, MATHIEU L. 4437 GUNNISON, 67220

55 M 3005 81 GS

HIRATZKA, TOMIHARU, 6 ST CLOUD PLACE, 67230

13 M 511 43 DO

HIZON, RAMON R. ST FRANCIS HOSP, 67214

262-6211

38 M 74801 62 DR

HOOSON, HERVEY R. 1122 S CLIFTON, 67218

03 M 1606 31 DO

HOLDEN JR, RAYMOND F. 262 SOUTH BROOKSIDE, 67218

10 M 2802 33 DO

HOLLAND JR, DAVID L. 2804 E FIRST, 67214

57 M 1902 84

HOLMES, JED. 7111 E 21ST, 67206

684-2851

53 M 3005 78 FP

HORBELT, DOUGLAS V. WESLEY MEDICAL ARTS BLDG, 67208

691-0251

47 M 4802 72 OBG

HOUSHOLDER, DANIEL FAIR, 7705 KILLARNEY CT, 67206

268-5910

43 M 1902 70 NM

HOUSHOLDER, MARTHA S. 835 NORTH HILLSIDE, 67214

685-4395

46 F 1902 72 D

HOWARD, DONALD D. 959 N EMPORIA, 67214

265-7241

11 M 1902 38 OPH

HUEBERT, DEAN A. 5025 E KELLOGG, 67218

682-1534

22 M 1902 46 FP

HULL, KENNETH L. 3429 E DOUGLAS, 67218

265-7903

38 M 2301 69 P

HULTGREN, MYRON K. 1520 S CLIFTON, 67218

685-1381

41 M 1902 68 FP

HJME, JOSEPH W, 5111 E 21ST,67208
 655-2223
 38 M 1902 69 DRG
 HUMMER, LLOYD M, 3244 E DOUGLAS,67208
 689-9323
 32 M 3901 57 1M
 HUND, LARRY R, 3333 E CENTRAL SUITE 408,67208
 682-0411
 52 M 1902 78 PD
 HUSTEAD, ROBERT F, 427 N HILLSIDE,67214
 683-7200
 28 M 801 54 ANES
 HUTCHINSON, STEVEN A, 4128 E ENGLISH,67218
 -
 59 M 1902 84
 HUTSEY, PAUL J, 1770 S ROCK RD APT 808,67207
 -
 49 M 1902 85
 HYNES, HENRY E, 1035 N EMPORIA,67214
 262-4467
 35 M 53902 58 HEM
 IBARRA, J LUIS, 1035 N EMPORIA,67214
 262-1853
 20 M 64901 46 3
 IDBELS, BADR, 1100 N ST FRANCIS STE 240,67214
 267-0159
 47 M 87501 72 TS
 ISAACS, JUANITA J, 3101 E 9TH,67208
 775-6209
 43 F 2101 P
 JACKSON, CHARLES R, 1035 N EMPORIA,67214
 263-0812
 27 M 1606 53 GS
 JAMES, DONALD L, 1301 N WEST,67203
 745-5245
 42 M 3901 71 DTD
 JAMES, VERNOY L, 3333 E CENTRAL SUITE 816,67208
 685-5326
 29 M 3601 55 PD
 JAWADI, JAMEELA MUSAIN, 3333 E CENTRAL SUITE 610,67208
 686-6659
 50 F 49521 73 PD
 JAZAYERLI, NABIL, 1152 S CLIFTON,67218
 681-2401
 44 M 87501 70 CD
 JEHAN, SAYED S, 635 N MAIN,67203
 268-8306
 33 M 70403 59 P
 JENNEY, CHARLES B, 818 N EMPORIA SUITE 200,67214
 263-0296
 34 M 2834 61 GS
 JENSEN, DARAN L, 3333 E CENTRAL SUITE 301,67208
 685-7234
 52 M 3005 79 DRG
 JESTER, SHELBY L, 818 N EMPORIA STE 307,67214
 268-6189
 43 F 4102 74 ANES
 JOHNSON, CAROL ANN, 3243 E MURDOCK SUITE 303,67208
 688-3070
 49 F 1902 77 FP
 JOHNSON, GEORGE K, UKSM WICHITA,67214
 268-5984
 40 M 1205 67 1M
 JOHNSON, THOMAS E, 3333 E CENTRAL,67208
 685-1291
 41 M 1643 67 R
 JOSEPH, JAPHET G, 1111 N ST FRANCIS,67214
 265-2613
 49 M 49531 74 CD
 JOST, GARY D, 212 N HILLSIDE,67214
 682-4572
 51 M 1902 77 GS
 JUDILLA JR, FRANCISCO, 2322 EAST CENTRAL,67214
 263-6186
 44 M 74801 71 ANES
 KADISON, HERBERT I, ST FRANCIS HOSP,67214
 262-6211
 44 M 1611 69 R
 KARDATZKE, E STANLEY, PO BOX 18008,67218
 943-9342
 39 M 1720 64 FP
 KARDATZKE, JON K, PO BOX 18008,67218
 943-3271
 36 M 1720 62 FP
 KASHA, ROBERT L, 1000 S WOODLAWN APT 302,67218
 682-1534
 11 M 2834 38 GS
 KASSEBAUM, KENNETH G, 3420 E DOUGLAS,67208
 685-6381
 34 M 1606 60 CHP
 KATER, ERIC DEAN, 550 N HILLSIDE,67214
 772-5609
 56 M 1902 82

KAUFMAN, EUGENE E, 3243 E MURDOCK,67208
 685-1491
 30 M 1902 56 DRG
 KEENE, GEORGE H, 5025 E KELLONG,67218
 682-1534
 20 M 1902 49 GS
 KELLER, JAMES P, 1431 S BLUFFVIEW STE 209,67218
 685-1284
 48 M 1902 74 1M
 KELLY, ROBERT W, 550 N LORRAINE,67214
 682-6511
 46 M 4802 72 DRG
 KENDALL, TOM E, 825 N HILLSIDE,67214
 688-7500
 37 M 3901 62 PS
 KENDRICK, J GILLERAN, WESLEY MEDICAL CENTER,67214
 688-2080
 20 M 1902 46 ADM
 KENNEDY, GERALD T, 16069 CAVALCADE LANE,67230
 594-5243
 35 M 1902 61 GE
 KEYES, MICHAEL J, 3101 E 9TH,67208
 684-0201
 44 M 2101 70 P
 KHICHA, GYANCHAND J, 818 N EMPORIA SUITE 200,67214
 263-0296
 37 M 49530 61 TS
 KHOURY, GEORGE H, 3333 E CENTRAL SUITE 416,67208
 681-2021
 32 M 33002 55 PD
 KIM, PAIK N, 818 N EMPORIA SUITE 403,67214
 262-4467
 33 M 58302 58 HEM
 KIMBLE, JAMES A, 3244 E DOUGLAS,67208
 689-9316
 45 M 702 71 OPH
 KIRIAKOS, LYNNE A, 2330 N OLIVER #919,67220
 -
 59 F 1902 85
 KIRK JR, E DAVID, 1431 S BLUFFVIEW DRIVE,67218
 685-1351
 34 M 1902 62 1M
 KISER, JOHN L, 3243 E MURDOCK,67208
 685-6222
 37 M 2802 62 GS
 KISER, WILLARD J, 3243 E MURDOCK,67208
 -
 05 M 4705 30 GS
 KITCHEN, ROBERT R, 3420 E DOUGLAS,67208
 685-2355
 26 M 1902 52 CHP
 KNAPP, LESLIE E, 302 S CRESTWAY,67218
 -
 96 M 1902 25 DD
 KNAPP, M ROBERT, 37 VIA ROMA,67230
 684-7251
 23 M 3519 47 ANES
 KNEIDEL, THOMAS W, 732 N TOPEKA,67214
 267-1924
 40 M 4101 66 DRG
 KNIGHT, LAURA C, ST FRANCIS REG MED CTR,67214
 268-5922
 42 F 502 68 DR
 KNIGHT, PHILIP J, 818 N EMPORIA SUITE 200,67214
 263-0296
 42 M 0502 68 PDS
 KOURI, SAMMY H, 3243 E MURDOCK,67208
 682-2911
 33 M 3901 57 GS
 KRAUSE, ROLAND L, 855 N HILLSIDE,67214
 685-1381
 25 M 1902 53 1M
 KREADY, JOHN L, 3243 E MURDOCK SUITE 300,67208
 685-8231
 48 M 1902 79 FP
 KRUPKA, JOHN J, 818 N EMPORIA #201,67214
 263-0348
 47 M 1642 73 VS
 KUBIN, SUSAN D, 1048 N DELLROSE,67208
 -
 59 F 1902 85
 KUBINA, GLENY RICHARD, MID-KANSAS ENT ASSOC,67211
 684-2838
 47 M 3840 72 DTD
 KURTH, C JOSEPH, 200 S ROCK RD SUITE H,67207
 687-6603
 10 M 3006 35 NP
 KUTILEK, FRANK J, 7200 W 13TH ST SUITE 3,67212
 722-4258
 30 M 1902 57 FP
 LAI, JENG Y, 959 N EMPORIA STE 205,67214
 265-2613
 41 M 38502 67 TS

LANCE JR. JOHN F. 3244 E DOUGLAS.67208
 689-9438
 20 M 1902 45 DRS
 LATIMER, KATHERINE. 3243 E MURDOCK SUITE 401.67208
 686-7327
 49 F 1205 75 ANES
 LAUVER, MARY ANN. 818 N EMPORIA SUITE 303.67214
 265-3774
 43 F 1902 74 PD
 LAWN, CLAUDIA A. 1035 N EMPORIA SUITE 285.67214
 264-2401
 50 F 1902 75 R
 LAWN, RAYMOND A. 715 N MISSION RD.67206
 683-8991
 09 M 2604 35 AM
 LEE, R REX. 6155 E HARRY.67218
 685-2306
 29 M 3901 55 FP
 LEE JR, EDWARD S. 2002 E 17TH ST.67214
 -
 09 M 4707 37 FP
 LEISY, JERALD W. 3310 E DOUGLAS SUITE 101.67208
 681-2937
 42 M 1902 68 P
 LEITNER, YORAM B. 3244 E DOUGLAS.67208
 689-9227
 53 M 3519 77 DTD
 LEVINE, WILLIAM R. UKSM-WICHITA.67214
 268-8388
 42 M 1902 67 P
 LIES, RICHARD B. 3244 E DOUGLAS.67208
 689-9111
 42 M 1902 68 RHU
 LIN, JDE J. 8630 HUNTINGTON.67206
 268-5420
 42 M 24404 69 PATH
 LINDSTROM, LORI A. 7007 E ZIMMERLY.67207
 -
 56 F 1902 85
 LINHARDT, RONALD D. 3243 E MURDOCK.67208
 683-2655
 36 M 2803 64 OBG
 LITTELL, JAMES A. 1520 S CLIFTON.67218
 685-2371
 44 M 1902 71 EM
 LITTLE, L GILBERT. 122 S WESTFIELD AVE.67209
 -
 99 M 401 27 P
 LIVINGSTON, PAULA T. D.O.. 2622 W CENTRAL.67203
 945-9161
 46 F 2879 77 EM
 LOCKHART, JOSEPH G. 5900 E CENTRAL.67208
 684-7239
 17 M 4113 43 PD
 LOEFFLER, JAMES A. 400 N WOODLAWN.67208
 685-5375
 36 M 3841 63 A
 LOEWEN, HENRY H. 2142 W 17TH.67203
 -
 03 M 1902 35 DO
 LOEWEN, WILLIAM C. 10300 W MAPLE.67209
 722-6892
 41 M 1902 71 FP
 LOGAN, GEOFFREY G. 212 N HILLSIDE.67214
 682-4572
 31 M 14303 56 OBG
 LDSEE, JOHN M. 3243 E MURDOCK STE 401.67208
 686-7327
 51 M 4301 77 ANES
 LOVE, ROBERT H. 348 S GREEN.67211
 -
 50 M 1902 86
 LDVETT, PAUL A. 110 PATTON.67208
 -
 09 M 1902 45 ORS
 LOW, HAROLD L. 1148 S HILLSIDE.67211
 684-2858
 18 M 1902 44 FP
 LUCAS, GEDRGE L. 3244 E DOUGLAS.67208
 689-9485
 34 M 1001 61 ORS
 LUDWIG, CARJ. S. 2280 S GLENDALE.67218
 -
 57 F 1902 84
 LUEKEN, LUEKE B. 3244 E DOUGLAS.67208
 689-9234
 23 M 40723 52 OBG
 LUELLEN, THOMAS J. 3244 E DOUGLAS.67208
 689-9244
 17 M 1902 41 IM
 LUZZATI, ENZO F. ST FRANCIS HOSPITAL.67214
 262-6211
 25 M 56119 50 R

LYGRISSE, DANIEL V. 1910 CHARLOTTE.67208
 688-2000
 50 M 64914 78 FP
 LYNCH, MARY A. 320 N HILLSIDE.67214
 682-3221
 48 F 1002 77 SM
 MADER, ELAINE M. 448 N FOUNTAIN.67203
 697-4431
 56 F 1902 83 FP
 MADISON JR, WARD N. 3600 E HARRY.67218
 686-9432
 37 M 3601 62 PATH
 MAGIDSON, ELLIOTT ARTHUR. 116 LONGFORD CT.67206
 686-2193
 43 M 1611 68 PATH
 MAGSALIN, ROMULO D. 1520 S CLIFTON.67218
 689-5775
 40 M 74808 60 GP
 MAILMAN, GERSHOM, ST JOSEPH MED CENTER.67218
 695-1111
 26 M 3519 49 ANES
 MANOELBAUM, MARK A. 3244 E DOUGLAS.67208
 689-9137
 53 M 3901 79 V
 MANNING, ROBERT T. UKSM WICHITA.67214
 688-2212
 27 M 1902 54 IM
 MANSOUR, BADIE S. 3243 E MURDOCK SUITE 401.67208
 686-7327
 45 M 33002 69 ANES
 MARSH, HENRY O. 818 N EMPORIA SUITE 306.67214
 262-7598
 18 M 1611 43 ORS
 MARTIN JR, GLEN E. 2322 E CENTRAL.67214
 263-6186
 20 M 1902 49 ANES
 MARYMONT JR, JESSE H. WESLEY MED CENTER.67214
 685-2151
 28 M 3515 54 PATH
 MASSEY, ANDREW D. 1001 N MINNEAPOLIS.67214
 268-8378
 50 M 1902 77 IM
 MASTID JR, GEORGE J. 3243 E MURDOCK.67208
 684-5235
 25 M 1902 52 GS
 MATASSARIN, BENJAMIN M. 2916 E CENTRAL.67214
 684-5243
 20 M 1902 45 IM
 MATASSARIN, FREDERICK W. 734 N EMPORIA.67214
 265-2382
 15 M 1902 37 U
 MAWDSLEY, MICHAEL W. 3333 EAST CENTRAL #610.67208
 686-6659
 49 M 1902 74 PD
 MCBOYLE, MARILEE. 818 N EMPORIA.67214
 263-0296
 52 F 1902 77 GS
 MCCLANAHAN, WARD A. 5105 E 21ST.67208
 684-8211
 22 M 3005 48 ORS
 MCCLELLAN, ERNEST L. 3243 E MURDOCK SUITE 401.67208
 686-7327
 38 M 4802 70 ANES
 MCCOY, C PATRICK. 3243 E MURDOCK SUITE 401.67208
 686-7327
 53 M 1902 79 ANES
 MCCOY, CHARLES P. 3333 E CENTRAL.67208
 685-7234
 17 M 3006 42 OBG
 MCCULLOUGH, JAMES P. WESLEY MED CENTER LAB.67214
 688-2810
 54 M 1902 79 PATH
 MCDONOUGH, W DAVID. 3244 E DOUGLAS.67208
 689-9111
 48 M 3305 76 U
 MCGUIRE, WILLIAM F. 3333 E CENTRAL.67208
 683-5655
 17 M 4101 43 PD
 MCMULLEN, BRUCE R. 1122 S CLIFTON.67218
 682-5012
 53 M 4002 79 IM
 MCQUEEN, DAVID ARNOLD. 818 N EMPORIA.67214
 262-7598
 47 M 64914 75 ORS
 MEEKER II, BRUCE P. 345 N HILLSIDE.67214
 686-3384
 30 M 1902 58 OBG
 MELEAN, JAIME. 1152 SOUTH CLIFTON.67218
 681-2401
 40 M 17602 65 CD
 MELHORN, J MARK. 656 S LORRAINE.67211
 268-5989
 53 M 1902 80 ORS

MELHORN, KATHERINE J. 656 S LORRAINE.67211
 268-8302
 55 F 1902 81 PD
 MENAKER, JEROME S. 2703 E CENTRAL.67214
 685-1277
 16 M 1002 41 DBG
 MENDOINES, L MARLENE. 835 N HILLSIDE.67214
 685-4395
 45 F 1611 70 D
 MENDOINES, RUPERTO D. 3243 E MURDOCK -SUITE 404.67208
 682-6585
 44 M 1611 71 1M
 MENEHAN, H JAMES. 3244 E DOUGLAS.67208
 689-9404
 26 M 1902 53 PD
 MENHUSEN, MCVY J. D.O.. 3243 E MURDOCK SUITE 401.67208
 656-7327
 48 M 1676 79 ANES
 MENKING, F W MANFRED. 3244 E DOUGLAS.67208
 689-9336
 34 M 40715 61 PD
 MENKING, SJSAN MARGARET. UKSM WICHITA.67214
 268-8302
 41 F 3840 67 PD
 MERCADER, MARID S. 2322 E CENTRAL.67214
 263-6186
 43 M 74801 64 ANES
 MEREDITH, W TOM. 1035 N EMPORIA.67214
 263-7285
 35 M 4812 61 1M
 MERRITT, JOE P. 2703 E CENTRAL.67214
 688-5211
 46 M 2802 71 DBG
 MERSHON, JAMES C. 933 N TOPEKA.67214
 263-5889
 37 M 1803 63 CD
 MESSANDRE, DEBRA L. 551 S DOPLAR.67211
 -
 58 F 1902 84
 MEYER, WARREN E. 1515 S CLIFTON SUITE 420.67218
 684-5237
 27 M 1606 51 GS
 MICHELBACH, ALBERT P. 2916 E CENTRAL.67214
 684-5243
 35 M 2101 61 1M
 MILFELD, DOUGLAS J. 818 N EMPORIA SUITE 200.67214
 263-0296
 45 M 4804 72 TS
 MILLER, DAVID PATERSON. 7111 E 21ST N.67206
 689-3070
 50 M 2803 77 FP
 MILLER, DON E. 4145 E KELLOGG.67218
 682-6551
 23 M 2802 44 GS
 MILLS, CHARLES D. 1140 S WATER.67213
 -
 89 M 2002 14 DD
 MILLS, PHILIP R. 3243 E MURDOCK #602.67208
 -
 49 M 512 76 PM
 MINNS, GAROLD D. UKSM-WICHITA DEPT OF MED.67214
 268-8378
 51 M 1902 76 1M
 MIRZA, MEOD. 3333 E CENTRAL.67208
 686-6683
 38 M 40733 64 PDS
 MODRE, DENNIS F. 1035 N EMPORIA.67214
 265-3226
 36 M 2101 62 HEM
 MORGAN, JAMES I. 3124 S SENECA.67217
 522-2266
 29 M 1606 53 FP
 MORGAN, RANDALL J. 212 N HILLSIDE.67214
 682-4572
 52 M 1902 77 DBG
 MORGAN III, LOUIS S. 8030 E KELLOGG.67207
 683-3811
 22 M 3901 48 FP
 MORRISON, RICHARD L. 1148 S HILLSIDE.67211
 684-3391
 42 M 1902 67 FP
 MORROW, THOMAS F. 3310 E DOUGLAS.67208
 685-1443
 21 M 5606 46 P
 MOSELEY, JACK E. 1120 S CLIFTON.67218
 682-4982
 25 M 3901 53 FP
 MOSIER, STANLEY JAY. 3243 E MURDOCK.67208
 685-8231
 42 M 1902 68 FP
 MUELLER, VERNETTE A. 1431 S BLUFFVIEW.67218
 684-3981
 17 M 2802 41 DBG

MUETH, JON D. 7111 E 21ST.67206
 684-2858
 53 F 2803 79 FP
 MULLINIX, JANICE M. 3333 E CENTRAL SUITE 533.67208
 687-5443
 47 F 3006 73 N
 MURPHY, BARRY L. 3243 EAST MURDOCK #500.67208
 684-0251
 45 M 1902 71 1M
 MURPHY, DUANE A. 3243 E MURDOCK.67208
 685-1491
 32 M 1902 65 JRS
 MURPHY, PATRICK L. 7700 E KELLOGG.67207
 687-4020
 55 M 3901 81 FP
 MURPHY, PAUL M. 3600 E HARRY.67218
 685-1111
 28 M 3006 51 R
 MURPHY, WILLIAM R C. 818 N EMPORIA SUITE 200.67214
 263-0296
 43 M 1611 68 TS
 MURRAY, KENT B. VETERANS MEDICAL CENTER.67218
 263-6131
 47 M 3901 73 1M
 NELLIS, STEPHANIE F. 3244 E DOUGLAS.67208
 689-9270
 53 F 1902 79 1M
 NELSON, GERALD D. 825 N HILLSIDE.67214
 688-7500
 34 M 1902 60 PS
 NELSON, RUSSELL ALAN. 3333 E CENTRAL.67208
 685-5271
 18 M 1902 45 PD
 NELSON JR, GUST H. 3600 E HARRY.67218
 685-1111
 23 M 1902 46 DR
 NESMITH, LESLIE W. 3333 E CENTRAL.67208
 683-5611
 40 M 1902 66 OPH
 NETHERTON, DAVID M. 7111 E 21ST.67206
 684-2851
 55 M 2803 81 FP
 NEWBY, JAMES P. 818 N EMPORIA SUITE 200.67214
 263-0296
 34 M 1902 59 TS
 NEWSOM, F CARTER. 3310 E DOUGLAS.67208
 685-1443
 19 M 1201 43 P
 NIELSEN, MARY L. WESLEY MED CTR LAB DEPT.67214
 688-2468
 47 F 1902 77 PATH
 NIXON, WILLIAM A. 3333 E CENTRAL SUITE 525.67208
 683-6622
 16 M 1902 44 GS
 NORMAN, BENJAMIN R. 713 S ELLIS.67211
 -
 56 M 1902 85
 NORRIS, ROBERT P. 3244 E DOUGLAS.67208
 689-9232
 17 M 1902 43 1M
 NORTH, DORIS G. 1148 S HILLSIDE.67211
 684-5257
 16 F 1902 47 FP
 NORTH, VICTOR. 1148 S HILLSIDE.67211
 684-5257
 18 M 1902 47 FP
 NORTON, ROBERT K. 3244 E DOUGLAS.67208
 689-9235
 32 M 1001 57 PD
 NOWLIN, NANCY S. 1001 N MINNEAPOLIS.67214
 268-8378
 47 F 1902 74 1M
 NUILA, RICHARD F. 3244 E DOUGLAS.67208
 689-9234
 50 M 34104 76 DBG
 O'DONNELL JR, LEDNARD A. 8033 E DOUGLAS.67207
 684-2835
 27 M 1902 55 1M
 O'NEAL, JON T. 3701 EDGEWOOD.67208
 -
 57 M 1902 85
 OCHSNER, BRUCE B. 1035 N EMPORIA SUITE 235.67214
 263-6273
 39 M 1902 65 DPH
 ODENHEIMER, BURTRAM J. 3244 E DOUGLAS.67208
 689-9137
 48 M 2105 73 N
 ORTH-BALMAN, DIANE M. UKSM-WICHITA.67214
 -
 56 F 1902
 OSBORN, J CLARK. 1518 HASKELL.67213
 -
 57 M 3901 83 FP

OSBORNE, CONRAD C. 855 N HILL SIDE, 67214
685-1381
38 M 1902 67 FP
OSIO, ANTONIO L. 1520 S CLIFTON, 67218
685-2371
41 M 26404 65 EM
OSORBA, WILLIAM G. 2525 W 13TH, 67203
943-9391
25 M 2802 51 FP
OUANO JR. 8181 AND 8. 1515 S CLIFTON SUITE 380, 67218
684-5094
40 M 74801 63 U
OWEN, LARJE W. 810 N LORRAINE, 67214
685-2207
19 M 1902 50 ANES
OWEN, PERE A. 1128 S CLIFTON, 67218
684-7251
37 M 1902 64 ANES
PAGE, RUTH. 1051 N STRATFORD, 67206
-
13 F 1902 43 OO
PALMER, DAVID L. 4805 W CENTRAL, 67212
945-5177
37 M 1902 63 A
PARK, ROGER WALTER. 3244 E DOUGLAS, 67208
689-9217
43 M 1902 69 PD
PARKER, HAROLD L. 7027 FARMVIEW CT, 67206
684-1599
32 M 1902 67 FP
PARMAN, CRAIG R. 2501 E STAFFORD, 67211
-
56 M 1902 84
PASSMAN, STEVEN M. 835 N HILL SIDE, 67214
685-4395
47 M 2803 73 D
PATTERSON, BRUCE W. 1520 SOUTH CLIFTON, 67218
685-2371
46 M 1902 73 EM
PATTON, J MICHAEL. 1431 S BLUFFVIEW STE 210, 67218
686-2111
51 M 3005 78 FP
PAXTON, EDWARD SCOTT. 3600 E HARRY, 67208
689-5675
51 M 2802 77 PATH
PAY, NORMAN T. ST FRANCIS HOSP, 67214
268-5914
45 M 74802 68 NR
PEERY, WILLIAM M. UKSM WICHITA, 67214
261-2650
46 M 4802 73 IM
PELLETIER JR. LAWRENCE L. UKSM WICHITA, 67214
268-8378
42 M 3501 68 IM
PENCE, CHARLES D. 3244 EAST DOUGLAS, 67208
689-9468
42 M 1902 68 ORS
PENNINGTON, KATHERINE. 2113 SO BLUFF CT, 67218
685-5271
16 F 1902 43 PD
PETERIE, JERRY P. 818 N EMPORIA SUITE 305, 67214
264-3505
49 M 1902 75 IM
PETERS, THOMAS J. 3244 E DOUGLAS, 67208
689-9190
47 M 2803 77 IM
PHAN, DUNG MY. 959 N EMPORIA S 28, 67214
267-5580
48 F 94101 75 FP
PHIPPS, JACK G. 315 N HILLSIDE, 67214
686-3391
21 M 1902 53 FP
PIBURN, MARVIN F. 125 N ZELTA, 67206
267-4201
22 M 1803 48 GS
PICKENS, ANDREW T. WICHITA PSY PO BOX 8037, 67208
684-0201
43 M 2834 69 P
PINSKER, JACOB A. 1035 N EMPORIA, 67214
-
06 M 1902 35 IM
PLUMMER, PATRICK M. 427 S PAULA APT D, 67209
-
59 M 1902 84
POLING, TERRY L. 6155 E HARRY, 67218
685-2306
36 M 1902 62 FP
POLLACK, SIMON. 7523 PLAZA LANE, 67206
-
M 36 OO
POLLANO, STEPHEN M. D.O., 1035 N EMPORIA SUITE 185, 67214
265-5291
39 M 2878 68 PM

POLLOCK, ANTHONY G A. 1035 N EMPORIA STE 140, 67214
264-2806
45 M 8305 71 ORS
POOLE, BERNARD T. 1035 N EMPORIA, 67214
264-2806
37 M 53902 62 ORS
PORTER, GARRY L. 3243 E MURDOCK STE 400, 67208
686-7351
35 M 1606 61 P
POWERS, K DEAN. 2703 E CENTRAL, 67214
685-1277
23 M 1902 47 ORG
PRIETO, LUIS E. 959 N EMPORIA #203, 67214
265-2613
39 M 26407 65 IM
PULLMAN, NORMAN K. 3007 E CENTRAL, 67214
686-7369
21 M 3006 45 PS
PULLUM, RICHARD W. D.O., 2622 W CENTRAL, 67203
945-9161
27 M 1875 57 R
PURINTON, LEW W. 1431 S BLUFFVIEW DR, 67218
- 685-1301
23 M 1902 48 IM
PUTNAM, LYLE B. 4700 W 13TH UNIT 1-1, 67212
-
11 M 1902 36 OO
RAGHAVAN, PARULA P. 2404 GREENLEAF CT, 67226
263-6131
47 F 49501 70 IM
RAGHAVAN, PRAKASH V. 1100 N ST FRANCIS STE 240, 67214
262-7662
46 M 49501 69 CO
RANDALL, GEDRGE R. MID-KANSAS ENT ASSOC, 67211
684-2838
43 M 2802 69 DTJ
RANDES, MICHAEL J. 959 N EMPORIA, 67214
265-0789
48 M 1902 73 IM
RAUSA JR. FRANCISCO C. 1148 SOUTH HILLSIDE, 67211
683-4658
42 M 74808 66 IM
RAWCLIFFE JR. ROBERT A. 732 N TOPEKA, 67214
267-1924
29 M 3501 55 ORS
RAZEK, MANA A. WESLEY MED CTR PATH DEPT, 67214
688-2810
47 F 33004 71 PATH
RAZEK, ZACK A. 818 N EMPORIA SUITE 200, 67214
263-0296
45 M 60501 70 COTS
READER, G WHITNEY, PO BOX 2517, 67201
263-5889
48 M 2101 70 CO
REALS, WILLIAM J. UKSM WICHITA, 67214
261-2600
20 M 3006 45 PATH
REAZIN, WALTER L. 1430 HOMESTEAD, 67208
685-1381
30 M 1902 58 FP
REDDI, RAGHUNATH P. ST JOSEPH MEDICAL CTR, 67218
685-1111
36 M 49521 64 RT
REED, A J. 1520 S CLIFTON, 67218
685-5775
40 M 3901 65 EM
REED, D CRAMER, WESLEY MED CTR HLTH STRAT, 67214
689-2080
15 M 2802 41 ADM
REED, DAVID D. 3333 E CENTRAL, 67208
685-1291
43 M 1902 69 OR
REED, WILLIAM RANDALL. 550 N HILLSIDE, 67214
688-2360
51 M 1611 77 NEO
REEVES, BRADFORD F. 3333 E CENTRAL, 67208
685-1291
37 M 4812 62 R
REISMAN, MICHAEL ALAN. 3243 E MURDOCK SUITE 600, 67208
683-5688
50 M 4804 75 JPH
RELIHAN, DONALD A. 655 N WOODLAWN, 67208
684-5158
27 M 1902 54 OPH
REMPEL, JOHN M. 1515 S CLIFTON SUITE 240, 67218
685-1812
38 M 3901 62 PS
RHODAS, JAMES P. 3244 E DOUGLAS, 67208
689-9106
34 M 3520 60 IM
RHODEN, CURTIS M. 3243 E MURDOCK, 67208
684-0252
33 M 1606 59 IM

RHODES, IVAN E. 3635 ELMWOOD, 67218

685-1291

25 M 3901 49 R

RHODES, LDWELL M. 315 N HILLSIDE, 67214

685-1461

25 M 1902 53 FP

RICHARDSON, STEWART F. 3420 E DOUGLAS, 67208

695-2321

28 M 3005 54 P

RIEDERER, ROBERT E. 1131 S CLIFTON, 67218

685-4354

16 M 1902 42 FP

RIEGER, ERNEST H. 3243 E MURDOCK, 67208

682-4591

29 M 1902 56 GS

RIEPE, ROGER E. WESLEY MED CENTER, 67214

699-2810

46 M 1803 74 PATH

RIGGS, PAUL A. 808 S LEXINGTON, 67218

-

54 M 80 GS

RIORDAN, HUGH D. 3100 N HILLSIDE, 67219

682-9241

32 M 5605 57 P

ROADCH, NEIL E. JKSM WICHITA, 67214

268-8388

38 M 1902 67 CP

ROAN, YEAL. PERINATAL DIVISION, 67214

698-2384

41 M 38501 67 PD

ROBERTS, DANIEL K. 3333 E CENTRAL SUITE 301, 67208

698-3185

36 M 3005 61 DRG

ROBERTS, ROGER W. D.O., 3010 W CENTRAL, 67203

945-5221

49 M 2879 75 CD

ROBERTSON, JOSEPH K. 818 N EMPORIA SUITE 200, 67214

263-0296

41 M 3901 66 GS

ROBINSON, G DONALD. 3333 E CENTRAL, 67208

686-6659

28 M 1902 54 PD

ROBINSON, JOHN E. 2708 E CENTRAL, 67214

686-7351

32 M 6201 50 P

ROBINSON, ROBERT H. 3244 E DOUGLAS, 67208

689-9445

20 M 1902 53 ANES

ROBL, DAVID A. 10300 W MAPLE, 67209

722-6892

48 M 1902 74 FP

RODRIGUEZ-RAMOS, ERNEST R. 1111 N ST FRANCIS, 67214

265-2613

42 M 27501 67 COTS

RODRIGUEZ-TOCKER, LILIA. 1111 N ST FRANCIS, 67214

265-2613

21 F 27501 49 IM

ROMALIS, BRIAN E. 3429 E DOUGLAS, 67218

682-5069

39 M 6201 63 P

ROOS, MAUREEN. 905 N EMPORIA, 67214

265-2876

53 F 1902 75 FP

ROSE, SHELBY D. 3333 E CENTRAL, 67208

681-2741

40 M 2012 68 PATH

ROSEN, DAVID. 818 N EMPORIA #303, 67214

265-3774

48 M 1902 74 PD

ROSENBERG, THOMAS F. 2627 E CENTRAL, 67214

684-0501

41 M 1642 68 A

ROSEN, ROBERT L. 3644 COUNTRY CLUB, 67208

-

50 M 1902 85

ROSS, DENNIS LEE. 1035 N EMPORIA SUITE 105, 67214

263-7285

47 M 3005 73 VEP

RUSSELL, PHILIP W. 3244 E DOUGLAS, 67208

689-9351

22 M 1902 44 IM

SABIN JR, GEORGE M. 707 N MAIN, 67203

268-0824

12 M 5002 39 ADM

SADIO, SULEMAN. 1144 N ST FRANCIS, 67214

267-2159

40 M 70401 63 TS

SAEED, MOHAMMAD. 1520 S CLIFTON, 67218

685-2371

42 M 70404 66 IM

SANTOSCOY, GILBERT S. 3244 E DOUGLAS, 67208

689-9124

38 M 4812 62 GS

SCHILTZ, FRANCES. 115 S RUTAN #108, 67218

-

93 F 6701 23 DD

SCHLACHTER, ERNEST R. 406 E CENTRAL, 67202

265-0705

24 M 1902 52 FP

SCHLICHER, JOHN E. 3244 E DOUGLAS, 67208

689-9346

40 M 1803 66 D

SCHLUETER, JOHN J. 3333 E CENTRAL, 67208

685-9289

31 M 3841 56 R

SCHLYER, ARTHUR M. 940 N TYLER, 67212

721-1111

51 M 1902 80 FP

SCHMIDT, KENLEY D. 2330 N OLIVER APT 1012, 67220

-

55 M 1902 85

SCHNELLE, JOACHIM. 4145 EAST KELLOGG, 67218

682-0621

44 M 40933 70 FP

SCHNPF, CLIFTON C. 425 E MURDOCK, 67214

945-0142

29 M 1902 57 FP

SCHWARTZ, V DEAN. 400 N WOODLAWN #4, 67208

594-3881

24 M 1902 48 FP

SCOTT, WILLIAM H. 1431 S BLUFFVIEW STE 111, 67218

685-1111

41 M 4901 65 CD

SEN SARMA, PRONAB K. 1144 N ST FRANCIS, 67214

267-0159

45 M 49518 71 CD

SHAFFER, PRESTON J. 3333 E CENTRAL, 67208

685-7234

20 M 3005 46 DRG

SHAH, MUKHTAR H. 3243 E MURDOCK STE 400, 67208

686-2994

40 M 70404 63 P

SHAW, RICHARD C. 825 N HILLSIDE, 67214

688-7500

35 M 1902 61 PS

SHEHI, LORA J. 834 CARTER, 67203

-

56 F 1902

SHELLITO, JOHN G. 3244 E DOUGLAS, 67208

699-9124

18 M 1606 43 TS

SHIELD, CHARLES. 818 N EMPORIA SUITE 200, 67214

263-0296

46 M 2802 72 GS

SHOFFNER, RICHARD W. 3244 E DOUGLAS, 67208

689-9271

53 M 3979 79 IM

SHRADER, C ERIC. 655 N WOODLAWN, 67208

-

47 M 1902 78 DPH

SHRADER, DOYLE A. 3333 E CENTRAL, 67208

592-4851

16 M 1902 41 EENT

SHURTZ, GLEN L. 3333 E CENTRAL SUITE 214, 67208

685-1291

40 M 4802 78 R

SIEGEL, ALBERT R. 3600 E HARRY, 67218

685-1111

22 M 1642 47 PM

SIEMENS, CHARLOTTE A. 430 BLAKE, 67213

-

60 F 1902 86

SIFFORD, R LAWRENCE. 959 N EMPORIA, 67214

265-0561

25 M 1803 52 IM

SIMMS, DAVID ALAN. 3244 E DOUGLAS, 67208

689-9422

50 M 3401 76 DR

SKIBBA, RICHARD M. 3244 E DOUGLAS, 67208

689-9477

43 M 5606 70 GE

SKOCH, MICHAEL G. 7677 E 21ST APT 904, 67206

-

57 M 1902 84

SLUTSKY, LAWRENCE JOEL. ST FRANCIS HOSPITAL, 67214

258-5922

46 M 3501 72 DR

SMITH, ALVIN L. 929 N ST FRANCIS, 67214

262-6211

28 M 5606 57 PATH

SMITH, MARK A. 339 N HOLYOKE, 67208

-

54 M 1902 84

SMITH, TIMOTHY WM. 1035 N EMPORIA STE 270, 67214

269-4026

49 M 1902 74 IM

SMITH JR, WILLARD J, 851 N HILLSIDE, 67214
685-1371
32 M 1611 57 U
SNYDER, GREGG M, 902 N HILLSIDE, 67214
685-2377
27 M 1803 54 NS
SO, OMJN, HERMAN, 835 N HILLSIDE, 67214
685-4395
37 M 2701 62 D
SOLTZ, ROBERT A, 3244 EAST DOUGLAS, 67208
689-9381
47 M 2803 74 PD
SOMERS, MARVIN M, ST FRANCIS HOSP, 67214
262-6211
23 M 1902 48 R
SPANN, RICHARD H, 3243 E MURDOCK, 67208
684-0252
40 M 1902 65 PUD
STAMPS, PHIL, ST JOSEPH MEDICAL CENTER, 67218
685-1111
37 M 3901 63 PATH
STANLEY, KENNETH E, 959 N EMPORIA, 67214
257-0256
31 M 1902 56 U
STARK, JAMES R, 3244 E DOUGLAS, 67208
689-9422
20 M 1902 44 R
STECKLEY, RICHARD ALLEN, 1035 EMPORIA SUITE 130, 67214
263-3271
49 M 2105 74 1M
STEIN, PAUL S, 3243 E MURDOCK, 67208
685-2377
40 M 3305 66 NS
STEMBRIDGE, TRAVIS W, 3333 E CENTRAL SUITE 301, 67208
685-7234
47 M 4802 76 D8G
STEPHANZ JR, GERALD B, 834 CARTER, 67203
265-5554
57 M 1902
STOJT, KEVIN B, 6000 MAINGATE APT 303, 67220
-
56 M 1902 84
STREET, DAVID E, 818 N EMPORIA SUITE 200, 67214
263-0296
35 M 2101 61 GS
STREIT, JEROME G, 1131 S CLIFTON, 67218
685-4354
48 M 1902 77 FP
STRYKER, TERRY MARGARET, 3243 E MURDOCK - STE 300, 67208
685-8231
47 F 1002 75 FP
SUERO, JESUS T, 1148 S HILLSIDE, 67211
681-3371
33 M 74802 57 PUD
SULLIVAN, CORNELIUS J P, PO BOX 8503, 67208
-
19 M 3509 43 DD
SULLIVAN, LEONARD L, 3244 E DOUGLAS, 67208
689-9454
35 M 1902 61 PD
SUMMERS, LAURIE K, 1520 N OELL ROSE, 67208
-
54 F 1902 86
SVDBDDA, LUIS V, 3243 E MURDOCK STE 300, 67208
685-8231
39 F 1602 66 FP
SVDBDDA, WILLIAM B, 3243 MURDOCK SUITE 601, 67208
685-2377
36 M 1602 63 PDN
SWARTZ, MARSHA A, 1945 N ROCK RD APT 904, 67206
-
44 F 1902 86
SWEET, DONNA E, UKSM WICHITA, 67214
268-8378
48 F 1902 79 1M
TARVER, STEPHEN D, 3513 N ATHENIAN, 67204
-
55 M 1902 85
TATPATI, DANIEL A, 1144 N ST FRANCIS, 67214
267-0159
44 M 49535 67 TS
TATPATI, OLGA ADELINA, UKSM WICHITA, 67214
268-5992
44 F 49535 67 PD
TAYLOR, RICHARD J, 929 N ST FRANCIS, 67214
262-1952
21 M 3006 49 PATH
THELEN, J CHRISTINE, 1738 N RODSEVELT, 67208
-
13 F 5104 37 DD
THOMPSON, DANIEL M, PO BOX 4044, 67204
838-3381
19 M 1902 50 FP

THOMPSON, WILLIAM E, PO BOX 8253, 67208
-
30 M 3005 55 DPH
TIMEN, EDWARD N, 3244 E DOUGLAS, 67208
689-9481
24 M 1606 48 1M
TIMEN, HENRY N, 1227 N RIVER BLVD, 67203
-
96 M 1601 19 DD
TILLER, GEORGE R, 5101 E KELLOGG, 67218
684-5255
41 M 1902 67 AM
TILTON, FRANK M, 3244 E DOUGLAS, 67208
689-9137
33 M 2002 59 N
TINKER, ROBERT C, 1126 S CLIFTON, 67218
682-7578
31 M 2834 57 DRS
TINTEROW, MAJRICE M, 3333 E CENTRAL, 67208
685-4389
17 M 4802 41 ANES
TIPPIN JR, ERNEST E, 959 N EMPORIA, 67214
265-5256
24 M 1902 50 DTD
TODDER, ALFRED M, 1111 N ST FRANCIS, 67214
265-2613
15 M 4802 40 COTS
TONY, GERHART R, 855 N HILLSIDE, 67214
685-1381
16 M 1902 44 FP
TODDHEY, JOHN S, 3244 E DOUGLAS, 67208
637-9175
50 M 5605 77 DRS
TOSH, FRED E, 1900 E NINTH, 67214
265-8391
30 M 4706 54 PH
TRACY, TERRY A, 3333 E CENTRAL, 67208
685-7234
35 M 2803 61 D8G
TRAVERS, HENRY, MESLEY MED CTR LAB MED, 67214
688-2812
46 M 4114 71 PATH
TRETBAR, HARVEY A, 3243 E MURDOCK #500, 67208
684-9251
25 M 1902 52 1M
TREWEEKE, MICHAEL W, 2916 EAST CENTRAL, 67214
684-5234
46 M 1902 72 1M
TRIMMER, KENNETH J, 2804 E FIRST, 67214
-
58 M 1902 85
TRUDEAU, DAVID L, ALCOHOL TREATMENT UNIT, 67218
685-1111
40 M 2604 66 ADT
TRUJILLO, ANTERO A, 3600 E HARRY, 67218
685-1111
36 M 73701 61 ANES
UHLIG, PAUL J, 3244 E DOUGLAS, 67208
689-9191
23 M 1902 57 PD
VAN LEEUWEN, GERARD J, UKSM WICHITA, 67214
261-2631
29 M 1803 54 NED
VIN ZANT, LARRY E, 1515 S CLIFTON SUITE 270, 67218
682-6532
10 M 1902 40 GS
VINE, DONALD LEE, 3244 E DOUGLAS, 67208
689-9240
39 M 511 66 CO
VINZANT, WHITNEY L, 1515 S CLIFTON SUITE 270, 67218
685-1991
45 M 1902 71 GS
VDRHEES, VICTOR J, 7701 E KELLOGG, 67207
681-1152
36 M 1902 68 FP
VDTH, DOUGLAS W, UKSM WICHITA, 67214
268-8378
34 M 1902 59 1M
WADE, EDWARD J, 216 S ERIE, 67211
686-6835
53 M 1902 80 ANES
WADUD, ABDUL, 1543 S HILLSIDE, 67211
682-6814
35 M 70409 60 P
WALKER, MARSHALL D, O.D., 1301 N WEST ST, 67203
945-5245
41 M 2878 72 DTD
WALLING, ADRIAN E, 1131 S CLIFTON, 67218
689-5500
47 M 80302 71 FP
WALLING, ANNE D, UKSM WICHITA, 67214
261-2607
47 F 80302 71

WARD, CYNTHIA L. 2330 N OLIVER APT 309.67220

58 F 1902 85
WARD, LARRY G. 818 N EMPORIA STE 307.67214
268-6189

54 M ANES
WARREN, LLOYD P. 5205 E 21ST.67208
683-7223
11 M 1902 36 OPH
WARREN, MARVIN L. 1128 S CLIFTON.67218

55 M 1902 80 ANES
WARREN, WIRT A. 2226 S MINNESOTA.67211
267-0520
09 M 2802 33 PGER
WARREN JR. JOHN W. 931 N YALE.67208

15 M 2501 39 00
WAXMAN, DAEL M. 1134 LEWELLEN.67203

58 M 1902 85
WEAVER, J ROBERT. 959 N EMPORIA.67214
265-5731

21 M 1902 48 FP
WEAVER, JACK D. 959 N EMPORIA.67214
265-7241
16 M 2802 42 OPH
WEBER JR. HUGO P. 1035 N EMPORIA.67214
263-7285

40 M 702 66 IM
WEBSTER, BOBBY W. 550 N LORRAINE.67214
685-0559
48 M 4802 74 OBG
WEIPPERT, EDWARD J. 10300 W MAPLE.67209
722-6892

44 M 1902 70 FP
WELCH, LAUREN K. 3243 E MURDOCK SUITE 601.67208
635-2377

35 M 1902 61 N
WELCH, MARTIN H. UKSM WICHITA.67214
268-8378

36 M 5605 61 IM
WELLS, MAX MICHAEL. 550 N HILLSIDE.67214
688-2820

50 M 1902 76 PATH
WELLSHEAR, CHARLES C. WICHITA PSY CENTER.67208
684-0201

30 M 4706 58 P
WENINGER, JOHN H. 1148 S HILLSIDE.67211
682-6523

32 M 3005 62 FP
WEST, WILLIAM T. 3244 E DOUGLAS.67208
689-9234

24 M 1902 49 OBG
WHALLON, JACOB T. 202 N ROCK RD APT 120.67206

14 M 1720 41 00
WHEELER, NICKY RAY. 1515 S CLIFTON SUITE 390.67218
684-0220

48 M 1902 74 PS
WHEELER, PINCKNEY R. 2208 W 13TH.67203
943-2118

18 M 3901 56 FP
WHITAKER, JAMES A. 3243 E MURDOCK.67208
684-0251

44 M 1902 72 IM
WHITE, CHARLES M. 3244 E DOUGLAS.67208
689-9422

15 M 3005 41 R
WIENS, TIMOTHY C. 2330 N OLIVER APT #1012.67220
683-0090

55 M 1902
WILDER, LOWELL W. 655 N WOODLAWN.67208
684-5158

35 M 4109 62 OPH
WILKINSON, LARRY K. 1520 S CLIFTON.67218
685-2371

46 M 1902 74 FP
WILLIAMS, BRUCE N. 905 N EMPORIA.67214
268-5996

54 M 4002 80 FP
WILLIAMS, CHARLES L. 3244 E DOUGLAS.67208
689-9268

16 M 2834 43 IM
WILLIAMS, TOD J. 1519 FAIRMOUNT.67208

53 M 1902 84
WILLIAMSON, STEPHEN K. VA MEDICAL CENTER.67218
685-2221

54 M 1902 79 IM
WILLNER, CATHERINE. 1542 FAIRVIEW.67203

54 F 1902 86

WINCHELL, H H FORSYTH. PO BOX 2579.67201
265-1684

34 M 3545 58 R
WINCHESTER, EUGENE B. 2601 E CENTRAL.67214
684-0271

18 M 3006 56 FP
WISDOM, JAY K. 15 LYNWOOD.67207

684-0271
12 M 1902 42 FP
WISNER JR. HARRY J. 3244 E DOUGLAS.67208
689-9169

17 M 3005 43 IM
WITTMANN, ALBERT F. 2323 N WOODLAWN.67220

10 M 2834 38 00
WOLFE, FREDERICK. 6611 E CENTRAL.67206
686-1152

36 M 3508 66 RHU
WOOD, GARY B. 3243 E MURDOCK #404.67208
684-2131

21 M 2802 45 IM
WOOD, ROBERT D. 2337 RUTLAND CT.67226
486-2127

26 M 1902 53 FP
WOODHOUSE, CHARLES L. 959 N EMPORIA.67214
265-8821

10 M 1902 34 ENT
WOODRING, CATHY S. 222 S RIDGE RD.67209
945-0142

51 F 3546 77 FP
WORSING JR. ROBERT A. 3244 E DOUGLAS.67208
689-9175

47 M 2604 72 ORS
WRAY, ALEXANDER J. 120 E 21ST ST.67214
838-4912

19 M 1902 49 FP
WRAY JR. REGINALD D. 3333 E CENTRAL SUITE 602.67208
685-4389

40 M 4113 66 ANES
WRENN, C J. 3244 E DOUGLAS.67208
689-9156

47 M 3005 73 A
WU, JIN-TZE. 3333 E CENTRAL SUITE 214.67208
688-2920

41 M 38502 67 TR
YEW, CLAIRE S. 3244 E DOUGLAS.67208
689-9327

54 F 2507 80 PD
YOCKEY, CHARLES C. 3243 E MURDOCK S 500.67208
684-0251

46 M 1902 72 IM
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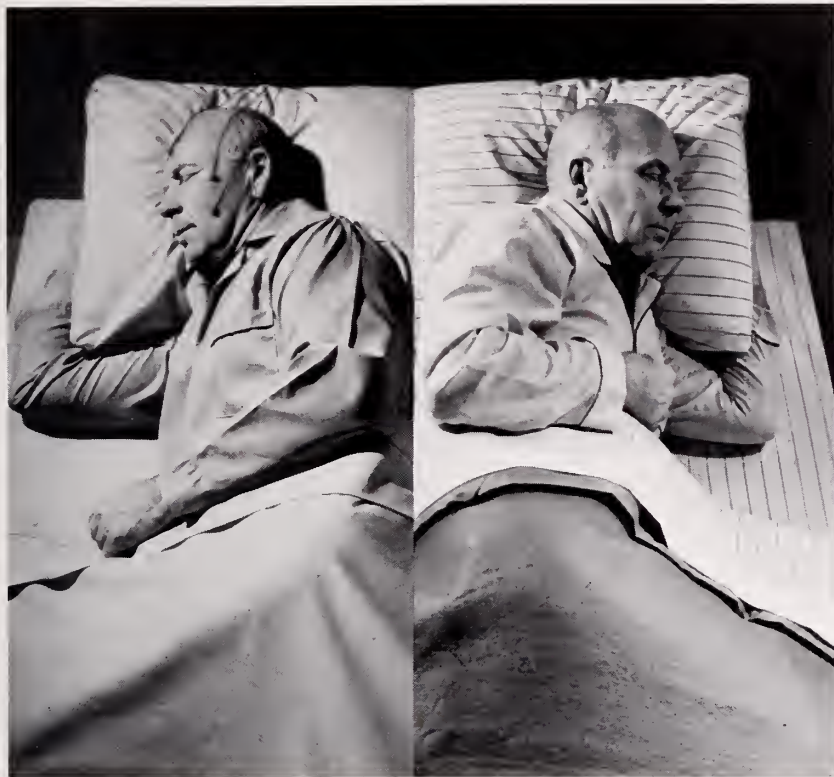
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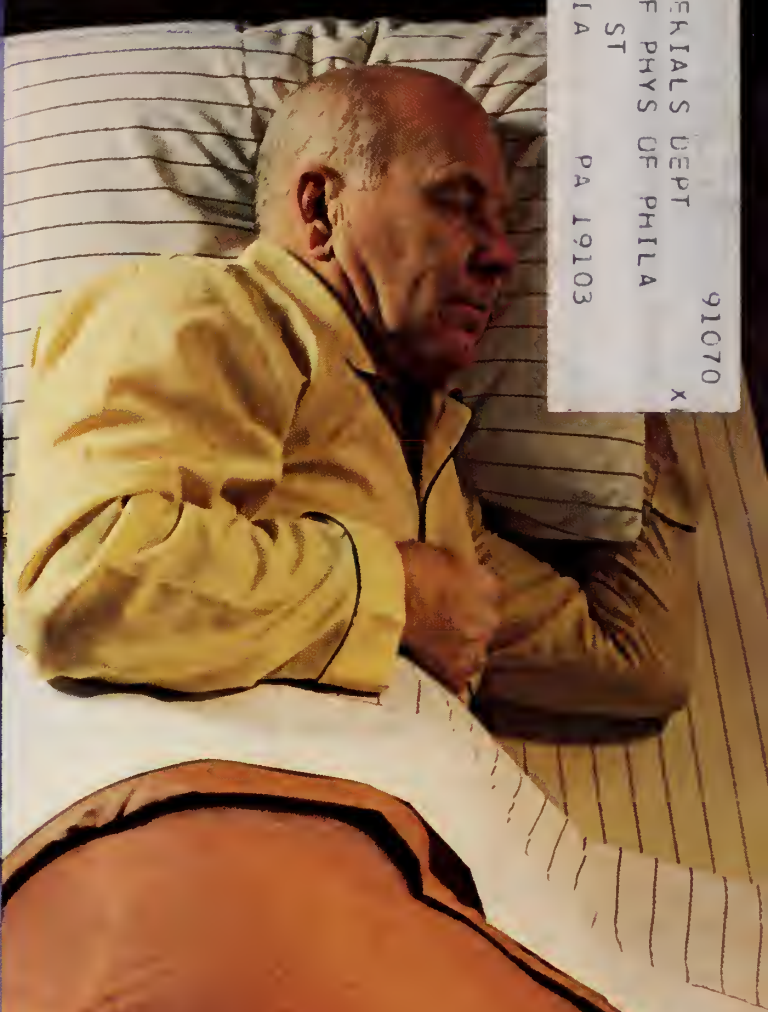
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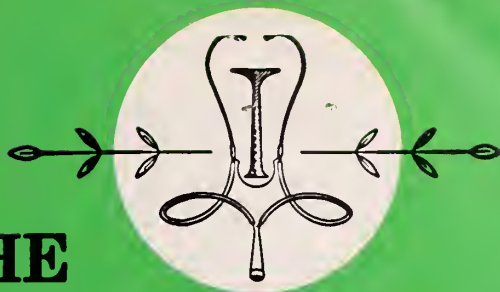
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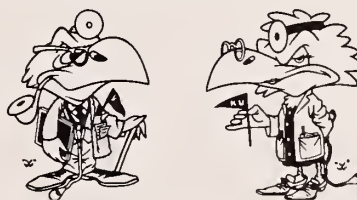
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1. Salzman C, Hoffman SA: Hosp Community Psychiatry 34: 897-902, 1983.
2. Ruffalo RL, Thompson JF, Segal JL: South Med J 74: 1075-1078, 1981.

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Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 125mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper G.I. disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia, some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chloridiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Transient amnesia or memory impairment has been reported in association with the use of benzodiazepines.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levartenerol Bitartrate Injection USP. Usefulness of dialysis has not been determined.

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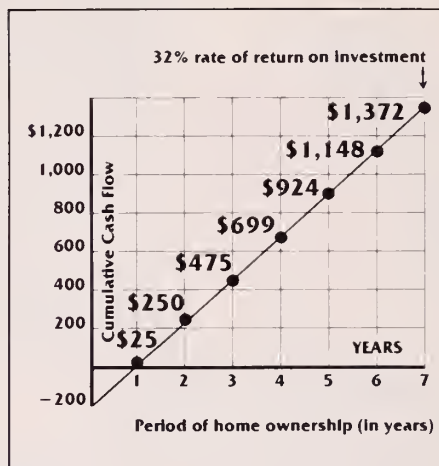
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Eleventh Annual UKSM-Wichita Issue

UKSM-Wichita: A Ten-Year Review

**WILLIAM J. REALS, M.D.* and
JANICE M. ARBUCKLE, M.A.,† *Wichita***

ON MAY 13, 1984, at the University of Kansas commencement exercises in Lawrence, the tenth class of medical students educated at UKSM-W received their diplomas. Since the school's inception in 1971, 353 physicians have completed their medical education in Wichita.

In 1971, the Kansas Board of Regents approved creation of a branch medical school in Wichita to be affiliated with Wichita State University, and two years later, the Kansas legislature appropriated the necessary funding.¹ The first 15 medical students to arrive in Wichita not only began a prestigious training program, but also were visible proof of a 38-year history of involvement in medical education by the University of Kansas in the Wichita area.

In 1946, the Wichita Veterans Administration Hospital became a member of The University of Kansas Medical Center VA Deans Committee.² With direction from UKSM, the VA Hospital in Wichita established residency programs in surgery, internal medicine and for a short time, radiology. Liaison with the programs at UKSM-KC was main-

tained through the Deans Committee and through the Sedgwick County Medical Society. Kansas City residents completed regular rotations in Wichita, and faculty from the Medical Center frequently visited to supervise and participate in these programs. Later, the medical residency was expanded to St. Francis and Wesley hospitals as the program grew.

The new school moved to succeeding larger quarters until by 1977 it had become apparent to administrators that sustained growth justified a separate facility. In June 1977, D. Cramer Reed, M.D., who was serving as Dean of both the WSU College of Health Related Professions and the medical school, resigned the WSU position to devote all of his efforts to faculty organization and the accreditation of the new University of Kansas School of Medicine-Wichita in the former Sedgwick County Hospital (E. B. Allen Memorial Hospital). Much is owed to Dr. Reed for his efforts to establish the school and to bring it through the formative process to its current high state of acclaim.

Each year, 50 students are selected by lottery from the sophomore class to come to Wichita for their junior and senior years. In most years, the number of students willing to come has outnumbered the positions available here. Clinical training is conducted in

* Dean and Professor of Pathology

† Associate Dean for Administration

Address reprint requests to Dr. Reals, UKSM-Wichita, 1010 No. Kansas, Wichita, KS 67214.

the three community hospitals (Wesley, St. Francis, St. Joseph) and the local VA Hospital with a total bed capacity of 2,500. In addition, students travel to various rural areas for preceptor training and rotations in primary care and rural medicine.* Preceptors at these sites hold faculty titles at our school.

A \$4.3 million renovation of the facility included a new outpatient clinic that opened in December 1983 to serve community health needs and to provide a training site for our medical students. Research laboratories for faculty and students are provided in the building as well as at the VA Hospital.

Retention

The state's support of resident training programs has proved quite effective for keeping Kansas medical school graduates in Kansas. Wichita hospitals and the medical school offer more than 225 residency training positions, of which 96 are supported through UKSM-W. Our medical school graduates' residency choices are widely distributed among the various specialties, but 58 per cent have chosen primary care.

Finance

A community-based medical school³ requires integration and interdependence within the medical community. Basic to this relationship is financing. State appropriations during the last ten years have grown from \$250,000 to more than \$6 million to meet the expanded education requirements as student population increased and residency programs flourished. As more faculty were recruited, additional contracts for professional services were signed with hospitals, and education and administration of medical services evolved. A valuable source of support for faculty salaries, contractual revenue reached almost \$1.3 million in 1984.

The varied sources of revenue to the medical school have allowed expanded operations beyond the original goal of training 100 medical students to include training programs in graduate medical education, health care outreach, continuing medical education, and biomedical research.

The medical school in Wichita has not only enhanced the quality of medicine, but has contributed

to the economic welfare of the area. Pass-through funds to the hospitals have never been less than 31 per cent of the school's state-appropriated budget. As USKM-Wichita prospers, the community benefits.

Support Services

To support faculty efforts, the medical school provides a variety of administrative services. A complete medical illustration service creates art work, slides, film, and teaching aids. In addition, the Division of Instructional Media maintains an interhospital color television microwave system which is used for closed-circuit TV conferences between the hospitals and the school. A small medical library is now in operation, but we look forward to the final phase of remodeling to provide a new improved library which will offer the most modern electronic links to access national library holdings and other data bases.

The Division of Postgraduate Education provides nearly 100 annual programs to meet the continuing medical education needs of faculty and area health professionals. There were 3,000 enrollments in these programs in 1983.

The Division of Health Care Outreach works closely with UKSM-Kansas City. A large hypertension screening program has served 144,000 people in the Wichita community since 1974. The Senior Health Program has served more than 2,000 senior citizens in the Wichita area since October 1981.

Summary

The school has made a major impact in health education as well as research and service in the Wichita area during its ten-year existence. Because we are a medical school without walls, we depend upon the community of Wichita, its health professionals, and hospitals to work with us in our teaching program. We are proud of our accomplishments in educating physicians and in serving the health needs of the public through our faculty and staff. We invite all members of the Kansas Medical Society to visit us and view the progress that has been made in medical education for the citizens of Kansas.

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2. Jones RAM, Healy PC, Perry JL: *A History of the Veteran's Administration Center, Wichita, Kansas*. Wichita, pp 5-6, 1983.
3. *Bulletin of The University of Kansas Medical Center* 20:1-2, 1970.

* Family Medicine preceptorship sites: Greensburg, Harper, Sterling, McPherson, Council Grove, Plainville, Hays, Wakeeney.

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Pediatrics preceptorship sites: Parsons, Hutchinson.

Departmental Reports

Department of Pediatrics

GERARD VAN LEEUWEN, M.D., *Chairman*

THERE HAVE BEEN a number of changes in faculty status during the last fiscal year while the house staff remains steady at nine. We now have four third-year, two second-year, and three first-year residents. Three UKSM-W graduates (Class of 1984) will join our residency at the PL-1 level for the 1984-85 year.

The junior clerkship has a one-month rotation at Wesley Medical Center and the remaining month at St. Francis Regional Medical Center or St. Joseph Medical Center. Ambulatory care has been expanded so that each student has one afternoon each week during the pediatric rotation. Students as a group spend one morning during their rotation at the Institute of Logopedics under the instruction of Bill Svoboda, M.D. The Pediatric Preceptorship Program is being developed. One student spent one month with Lynn Casey, M.D. in Hutchinson during the 1983-84 academic year. There are eight to ten students in pediatrics on each rotation, which is an ideal number for the amount of clinical material to teach pediatrics.

The 1984 senior students finished well above the national mean on the national boards examinations.

Several research developments have taken place under the direction of Richard Hoffman, Ph.D. The preliminary groundwork has been laid for establishing a pediatric/diabetes research laboratory during the UKSM-W Phase III renovation. In addition, a research committee has been established for the Wichita campus with Richard A. Guthrie, M.D. and Dr. Hoffman as representatives from the Pediatrics Department. The committee has generated \$30,000 annual seed grant funding, a semi-annual visiting lectureship program, and an internal funds program for the purchase of equipment used for research.

This school has seen considerable CME activity this year, including our co-sponsorship of the Governor's Conference on the Prevention of Child Abuse and Neglect; Culture Shock; Issues in Health Care of Indochinese Refugees, and New Diseases/Old Diseases Revisited.

Department of Family and Community Medicine

EDWARD P. DONATELLE, M.D., *Chairman*

THE DEPARTMENT of Family and Community Medicine has continued its growth and development during the past four years. Its administrative structure and faculty requirements are now well established to meet the needs of family medicine at UKSM-W. The department has accepted responsibility in the academic areas of scholarly activities, research, and service. Currently, department faculty consists of 17 full-time, 24 part-time, and 60 volunteer physicians from Sedgwick and neighboring counties.

The curriculum for undergraduate students consists of a one-month clerkship during the junior year and a one-month rural experience during the senior year. Elective courses are offered in community medicine and family practice. Attempts will be made to increase the curricular time for implementation of geriatric and community medicine as a component of our curriculum.

Students in their third year gain experience with family physicians in the Wichita urban area and also in our family practice residency centers at Wesley, St. Joseph, and St. Francis Hospitals, Wichita, and in Salina. A one-month rural experience is now offered at eight rural teaching sites in Harper, Greensburg, McPherson, and Sterling.

There are now 75 residents in training in the four divisional family practice residencies. We have had excellent results in recruiting Kansas students to our residencies and in retaining our residents in Kansas and the immediate perimeter area. During the last five years, 61 per cent have remained in Kansas to establish practice.

Continuing Medical Education programs for the practicing physician have been emphasized, and the department has worked closely with the Kansas Academy of Family Physicians to identify specific

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Ovarian Tumors in a Teenage Primigravida

ANNE D. WALLING, M.D.*; HENRY TRAVERS, M.D.† and
ADRIAN E. WALLING, M.D.‡, Wichita

OVARIAN FIBROMA and thecoma are seen less commonly in pregnancy than other tumors. This probably reflects the age distributions of the neoplasms which are skewed toward postmenopausal age groups. The relative frequencies of fibroma and thecoma, irrespective of age, are difficult to establish due to a broad overlap between the taxonomy of these two tumors. The pregnancy reported here was complicated by the simultaneous presence of fibroma and thecoma in which there were unambiguous physical and microscopic distinctions between the tumors.

Case Report

An unmarried 18-year-old Caucasian primigravida presented at 14 weeks' gestation. Physical examination and routine laboratory studies were normal. Examination at 27 and 36 weeks' gestation showed a normally progressing intrauterine pregnancy with a posteriorly positioned fetal head. At 42 weeks' gestation there was suspicion that the palpable posterior mass was not the fetal head, a fact confirmed by sonography and pelvimetry. A large, calcified soft tissue mass occupied the anterior pelvis. The patient was delivered by Caesarian section of a 3555 gram live, mature, female infant. The left ovary was incarcerated in the pelvis and contained two masses. The right ovary contained three masses. The left ovary and oviduct and the three masses in

the right ovary were removed. The patient tolerated the surgery well and subsequently made a complete recovery.

Pathologic Description

The large mass in the left ovary (*Figure 1*) weighed 450 grams and measured 9 x 9 x 7 cm. It was gray-white, smooth, and rock hard. The cut surface was trabeculated, gray-white to light yellow, and extensively calcified. An adjacent pedunculated mass measured 6 x 5 x 1.7 cm and was gray-white and rubbery. Microscopically, the large mass revealed interlacing, patternless fascicles of spindle-shaped cells associated with extensive sclerosis and calcification (*Figure 2*). There was a thin rim of

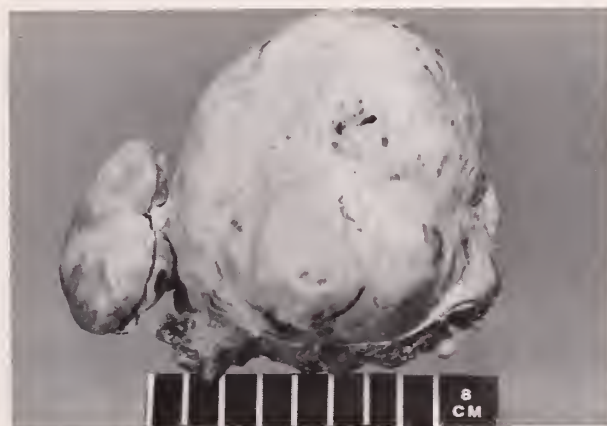


Figure 1. The large, focally calcified fibroma and the adjacent, smaller thecoma are connected by non-neoplastic fibrous tissue. Notice the surface bosselations of the larger mass which clinically mimicked the fetal head.

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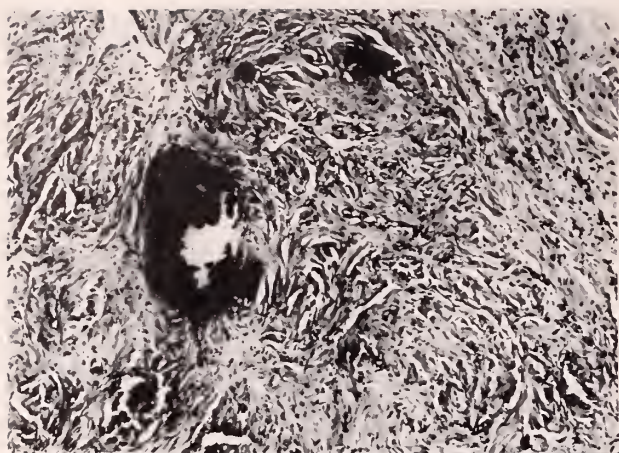


Figure 2. The fibroma is characterized by irregular fascicles of dense fibrous tissue with irregular areas of stromal calcification. (H & E x 100)

remaining ovarian cortex. Within the variably fibrous stroma of the pedunculated mass there were aggregates of polygonal cells (Figures 3a, 3b).

The right ovarian masses were of two types. One was an involuting corpus luteum while the other two were ill-defined stromal neoplasms composed of cords and small nests of polygonal cells identical with those in the pedunculated mass in the left ovary (Figure 4).

The hard left ovarian mass was interpreted as a fibroma, while its pedunculated partner was interpreted as a thecoma. The poorly defined right ovarian stromal masses were also considered thecomas.

Discussion

Although some authors consider fibroma and thecoma as related tumors representing opposite ends of a single spectrum of neoplasia,¹⁻³ the physical and microscopic differences between the two tumors in this case were so marked as to justify their interpretation as separate pathological entities. The large mass consisted of dense collagenized tissue with large areas of calcification arranged in a pattern that, when clinically palpated, mimicked the bones and sutures of a fetal head. Areas of calcification occur in less than 10 per cent of fibromata of the ovary, and the bone hard consistency in this case is unusual. The smaller tumor was a soft spongy mass with the characteristic luteinized cells of thecoma possibly showing exaggerated leuteinization due to the high human chorionic gonadotropin levels of pregnancy. Areas of sclerosis in this smaller tumor were suggestive of the sclerosing stromal tumor first described by Chalmardjian and Scully in 1973.⁴ This tumor is associated with women younger than 30 years and has been described as an incidental finding in pregnancy;⁵ however, this case lacked pseudolobulation and the mixed cellular architecture characteristic of sclerosing stromal tumor.

To date the simultaneous occurrence of fibroma and thecoma have not been recorded in a pregnant patient. Information on ovarian tumors in pregnancy is available from several large retrospective studies of hospital obstetric case records,⁶⁻⁸ but comparisons are limited by the different taxonomies used to classify tumors. In addition, individual studies have adopted different minimum size criteria for inclusion. For example, Tawa⁹ considered only tumors

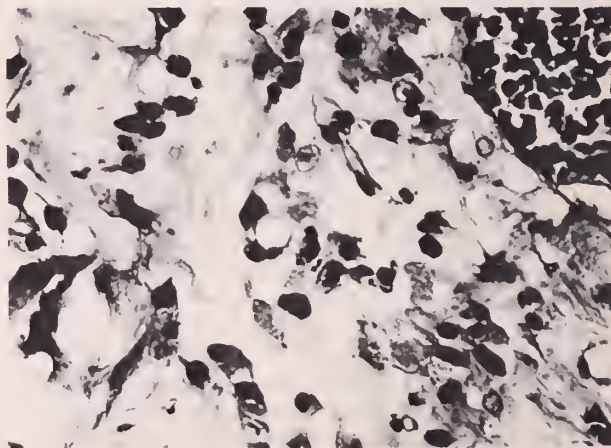
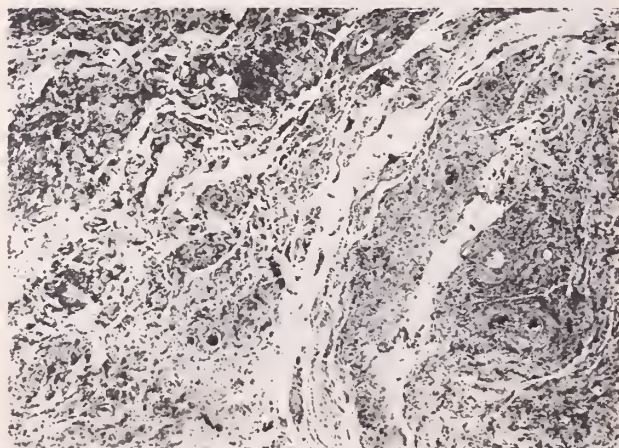


Figure 3. The thecoma is faintly nodular and is composed of loose fibrous tissue (left) within which are nests and cords of theca cells having granular to vacuolated cytoplasm (right). (H & E x 250)

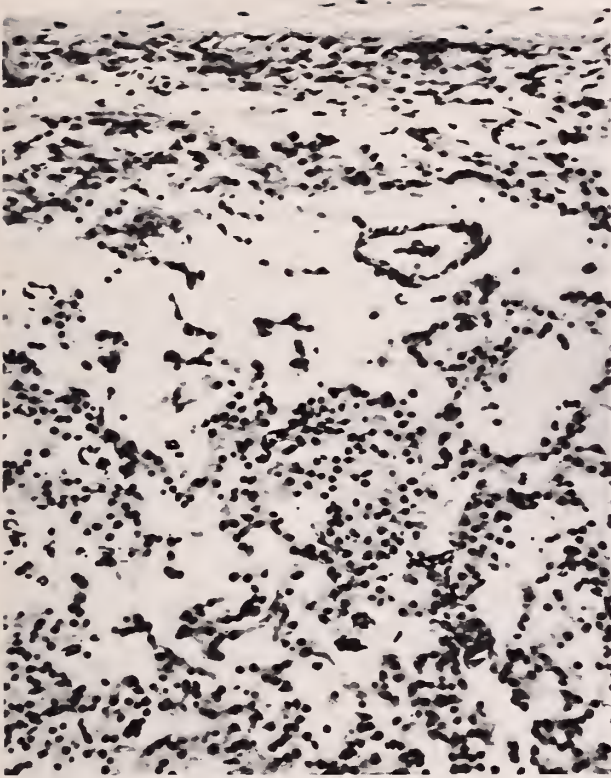


Figure 4. One nodule from the opposite ovary contained nests and cords of theca cells set within an edematous stroma. (H & E x 125)

with a diameter 6 cm or greater whereas Grimes¹⁰ included "cystic enlargement of any degree." Finally, there are differences between studies in the requirement to include only surgically and pathologically proven neoplasms. Thus, the reported incidence of any type of ovarian tumor in pregnancy varies from 1 in 81 to 1 in 2,300.⁹

In all reported series of ovarian tumors in pregnancy, cystic forms predominate and solid tumors

such as fibroma and thecoma are rare. In a review of his own and five earlier series in 1962, Chowdhury⁶ found only four solid tumors in 207 cases of ovarian neoplasm in pregnancy. More recently, Buttery⁷ *et al.* in Australia and White⁸ in the United States have confirmed the rarity of ovarian fibroma in pregnancy (Table I). Thecoma in pregnancy is even rarer.^{11, 12} Gough reported only 12 cases in the British and American literature by 1973¹³ to which must be added the case in Buttery's series⁷ reported later that year, plus Gee and Russell's case reported in 1981.¹⁴ Undoubtedly under-recognition and under-reporting contribute to this extreme rarity. Under-recognition reflects the belief that tumors of the fibroma-thecoma type do not occur in young women, but a study based on the files of the Armed Forces Institute of Pathology¹⁵ found that 8.3 per cent of all ovarian neoplasms in women aged 15-19 years were of the fibroma or thecoma type. Under-reporting is understandable as many tumor or cancer registries are restricted to malignant neoplasms so the only mechanism for measuring the incidence of other tumors is analysis of institutional records or individual case reports. Based on two of the largest series currently available⁷⁻⁸ (Table I), the incidence of fibroma in pregnancy could be 1 in 44,838 pregnancies and thecoma 1 in 179,351. If the tumors are indeed independent entities, their simultaneous occurrence in pregnancy would be almost unique.

Patients who have recorded cases of thecoma in pregnancy have experienced high mortality and morbidity attributed to tension, rupture, or infection of the tumor, or as a consequence of obstructed labor. Geogh¹³ reported three maternal deaths in 12 cases and only four surviving children (including one with severe brain damage) of the seven whose fetal outcome was known. Similar complications could be anticipated from a fibroma, but as no series has been recorded risks cannot be estimated.

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TABLE I
OVARIAN TUMORS IN PREGNANCY

Study	Obstetric Patients	Total Ovarian Tumors	Fibroma	Thecoma
Buttery <i>et al.</i>	153,890	164	3	1
White	25,461	37	1	0
Chowdhury	52,800	24	0	0

Experimental Approach to Diabetic Obesity

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DIANA SPEELMAN, M.A. and OLGA A. TATPATI, M.D., *Wichita*

THE MANAGEMENT of the obese patient with type II diabetes mellitus is a common therapeutic challenge. It is estimated that 80 per cent of type II, or adult onset, diabetic persons are obese.¹ Reduction in body weight has been reported to improve considerably the glucose tolerance in overweight persons with diabetes, and many persons will revert back to normal glucose tolerance after only a modest reduction in weight.² However, diet is often one of the most difficult aspects of the treatment program. A patient is often given a preprinted diet sheet to follow and told to lose weight. If the patient does not follow the "diet prescription" and shows no weight loss, s/he is considered a noncompliant patient and as a result of a feeling of guilt and failure, often drops out of treatment. It is not difficult to understand why many physicians consider diet therapy of diabetes a failure.³

Many patients who have failed to lose weight have used fad techniques for weight loss such as sensational diet plans, shots, pills and unscientific treatments. Often patients have not received intensive education in diet therapy. The goal of the diabetes team at the Kansas Regional Diabetes Center at UKSM-W is to educate the patient in skills that aid in understanding and treating diabetes. The Type II Diabetes Education Class has been established for that purpose. The class meets each month for two days, six hours each day, and can accommodate up to seven patients per class. The cost of the program is \$60, which at this time is not reimbursed by third party payment. Patients participating in the class are encouraged to bring a spouse or support person at no extra charge except for meals. Patients are scheduled for monthly followup visits as a group, and are also seen individually.

The overall goals of the class that pertain to each person with diabetes are to: normalize the blood glucose levels; attain healthy body weight; and prevent complications of diabetes. Through focusing on weight control, the goals can usually be met. To meet these goals, the Type II Diabetes Education

Class emphasizes weight control. Psychological needs, nutrition education, stress management, and physiology are presented to the participants in an effort to help them achieve their goals.

The program's diabetes counselor begins the class by presenting positive techniques for the diabetic to use to take control of the disease, to manage individual needs, and to make realistic plans and goals. Patients' feelings about themselves and their relationships often interfere with attempts to adhere to a diet. Consequently, people frequently indulge in food out of loneliness, boredom, anger, fatigue, depression, anxiety, and feelings of deprivation. Binging often occurs, and persons may begin to feel victimized by their eating patterns. The psychological portion of this class is aimed at helping participants to feel mastery over food and in control of their lives.

Development of realistic plans for achievement is an important element for success. Participants are instructed in making simple realistic plans that will fulfill needs for feeling good about themselves, foster good relationships, and to put more fun in their lives.

The nutrition coordinator presents the nutrition information vital to a successful weight loss program. Calorie needs are assessed and individual meal plans are prescribed and tailored to each patient's lifestyle. Calories are controlled through the use of calorie points, which are a simple but effective method of meal planning. One calorie point equals 75 calories. Patients learn the number of calorie points they are allowed for each meal and snack and choose foods accordingly. During the class, food is served cafeteria-style, and each patient plans his/her menu, chooses food, and weighs the portions on gram scales.

Nutrition education includes learning calorie point values of foods, weighing portion sizes, and basic eating guidelines for good nutrition. Basic eating guidelines include the avoidance of sugars, fat control, distribution of calories throughout the day, fiber intake, and nutrient information for planning healthy menus. Menus are planned for home use and time is allowed to discuss behaviors that interfere with weight loss and goals for overcoming

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self-defeating eating habits. Exercise is discussed; breaks are planned to include exercise and plans for exercise are discussed.

The program is designed to allow persons with diabetes to live as normally as possible in all situations. Travelling is discussed and calorie point lists for fast foods are distributed. Guidelines are presented for selecting from a menu when dining out. Reading labels on packaged foods and calculating calorie points in recipes are incorporated into the nutrition education.

The team's physician's assistant and diabetes program coordinator present information on anatomy and basic physiology so that the patient with diabetes learns how the body reacts to increased blood glucose levels, which may lead to complications of diabetes. Information is also presented on stress management, as stress is a factor in diabetes management.

A team can provide an educational service that is almost impossible for physicians to provide due to time restraints. The type II program was begun in December 1983, and 24 patients have participated.

Preliminary results are encouraging. The first followup visit six weeks after the December class showed an average weight loss of 6.8 pounds for the four participants. The followup class in February showed an average weight loss of 4.8 pounds for four participants. The March group averaged a weight loss of 7.3 pounds per person.

The approach to treatment of the patient with type II diabetes as described is a very intense educational program with an emphasis on behavior and attitudes. Patients are encouraged to assess current habits, modify as necessary, and learn attitudes and habits for success in weight loss and management of diabetes.

References

1. Smith M, Levine R: Obesity and diabetes. *Med Clin North Am* 48:1387-1397, 1964.
2. Rendell M, Ross DA, Drew HM, Zarriello J: Endogenous insulin secretion measured by C-peptide in maturity-onset diabetes controllable by diet alone. *Arch Intern Med* 141:1617-1622, 1981.
3. West KM: Diet therapy of diabetes: An analysis of failure. *Ann Intern Med* 79:425-434, 1973.

ROSTER CORRECTIONS AND ADDITIONS

Some corrections and additions to the information in the August (Roster) issue of the JOURNAL have come to our attention.

The Genetic Counseling Center listing for Wichita on page 30 should read as follows:

Wichita — Prenatal Diagnosis & Genetic Clinic, Division of Perinatal Medicine, Wesley Medical Center/UKSM-Wichita, 550 No. Hillside, Wichita KS 67214 — 316/688-2360

Wichita — Genetic Clinic, Department of Pediatrics, UKSM-Wichita, 1010 No. Kansas, Wichita KS 67214 — 316/261-2622

On page 28, the following addition should be made to the section on Poison Control Centers:

Topeka — St. Francis Hospital — 913/295-8095

The JOURNAL regrets these errors and any resulting inconvenience.

Purpura Fulminans

BARRY T. BLOOM, M.D.;* SECHIN CHO, M.D.† and
MEDO MIRZA, M.D.,‡ *Wichita*

PURPURA FULMINANS is an acute overwhelming illness, seen almost exclusively in children, in which early detection and therapy may decrease mortality and morbidity.

Case Review

A 20-month-old white male was brought to the hospital by air transport. He had been in good health until two days prior to admission when he developed a temperature of 39.4C and was noted to be drowsy. The following day he had a generalized tonic-clonic seizure. Examination revealed a lethargic child with otitis media, pharyngitis, and a rash. Vital signs were not available for review. He was admitted and placed on phenobarbital and Ceclor.

The patient was transferred to a Level II center after 16 hours. Evaluation there revealed a lethargic, but responsive child with circumoral cyanosis, peripheral mottling, a temperature of 38.3C, pulse 120/min, and respirations 48/min. No documentation of blood pressure was available for review. Urine output could not be differentiated from stools which were large in volume, free of blood, but foul-smelling. Vomiting began, so intravenous fluids were infused. Lumbar puncture revealed no evidence of meningitis. He continued to deteriorate and was subsequently brought into the authors' care.

The patient's past medical history was unremarkable. No recent exposures to drugs, toxins, or infections were known, and immunizations were up to date. Development was within normal limits by history.

The patient's admission temperature was 39.6C; pulse 132 and thready; blood pressure, 56 systolic; weight, 14.4 kg; and height, 91 cm. He was unresponsive to verbal stimuli, and painful stimuli resulted in decorticate posturing. His pupils were 2-3 mm in diameter and reactive to light, corneal reflexes were intact bilaterally, doll's eyes were not present, and he had gasping respirations.

Other pertinent physical findings included poor peripheral perfusion, swollen edematous extremities with purpura of hands and feet, bilateral exudative

conjunctivitis, tympanic membranes which were erythematous, but mobile bilaterally, an exudative pharyngitis, but no remarkable adenopathy in the cervical region. Desquamation was noted in postauricular region bilaterally.

Arterial blood gas tests revealed severe metabolic acidosis with adequate oxygenation (pH, 7.12; PCO₂, 21 mm Hg; HCO₃, 6.8 mEq/l) on ventilatory support. CBC revealed hemoglobin, 13.0 mg/dl; WBC, 14,800 cells/cu mm with a left shift and decreased platelets by estimation. Testing for electrolytes revealed a sodium, 122 mEq/liter; potassium, 6.6 mEq/liter; chloride, 101 mEq/liter; and bicarbonate, 6.1 mEq/liter. Blood sugar was 119 mg/dl; BUN, 58; creatinine, 2.0 mg/dl; SGOT, 321 U/liter; and calcium, 5.9 mg/dl ionized. Arterial ammonia was 94 mcg/dl. PT was 40 sec.; PTT, 90 sec.; fibrin split products, 1:20; and fibrinogen, 50 mg/dl.

During the first six hours after admission to this service, the patient required approximately 50 cc/kg of fluid for stabilization. Dopamine was utilized briefly to encourage renal blood flow.

Tagamet, phenobarbital, ampicillin, chloramphenicol, and prednisone were started. On the third day, heparin therapy was instituted, and CT scan revealed normal ventricular size without edema or hemorrhage.

On day five, the patient developed acute supratentorial herniation symptoms and, although attempts to salvage were made, he continued to deteriorate and died. Autopsy revealed epidermal necrosis with fibrin thrombi occluding blood vessels, and endothelial swelling. Periaqueductal fibrosis was noted with hydrocephalus, cerebral edema, and evidence of tentorial herniation. Diffuse fatty change of the liver was also noted. All cultures for viruses and bacteria performed in this hospital were negative for pathogens. Center for Disease Control tissue studies for rickettsial fluorescent antibody were negative. Only the initial nasopharyngeal culture in the primary hospital grew a pathogen, and it revealed *Hemophilus influenza*.

Case Discussion

Differential diagnosis of the generalized purpuric exanthem with a shock-like state should include systemic infections such as bacterial sepsis, rickettsial

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illnesses, and viral illnesses. Drug reactions with anaphylaxis or Stevens-Johnson syndrome may present in shock with purpura. Toxic shock syndrome and hemorrhagic shock and encephalopathy syndrome¹ should also be considered. Henoch-Schonlein purpura may be considered; however, systemic prodromal manifestations are usually noted, and marked volume depletion is rare in Henoch-Schonlein purpura. Dopamine-induced acral ischemia and infarction has been reported,² and this should be considered in any patient who has taken a sympathomimetic drug prior to the development of lesions.

This patient's autopsy findings, clinical course, and laboratory findings *may* suggest the diagnosis of Reye's syndrome; however, purpura fulminans, to our knowledge, has not been reported previously in association with Reye's syndrome.

Purpura Fulminans

Purpura fulminans, originally described in 1884 by Guilliot,³ is a catastrophic febrile illness with massive ecchymosis and intense systemic manifestations of chills, vomiting, occasional diarrhea, and prostration. The typical clinical picture is septicemia, shock, and disseminated intravascular coagulopathy.

Hjort, Rapport, and Jorgensen⁴ state that purpura fulminans should be defined as follows:

- It is a disease of children.
- It is preceded by a benign disorder, usually an infection. (Reports associate meningococcus,³ scarlet fever,⁵ measles,⁶ and varicella.⁷)
- After a variable but definite latent period, bleeding into the skin begins, spreads rapidly, and often progresses in waves.
- If the child survives long enough, the more extensive lesions undergo necrosis.
- Coagulation abnormalities compatible with disseminated intravascular coagulation are demonstrable.
- Histopathologic examination of the affected sites reveals widespread thrombosis of capillaries and venules.

The skin lesions are usually symmetrical and occur more often on the lower extremities. They begin as a port wine-colored lesion and progress to blood or serosanguinous filled bullae. Gangrene follows and the eschar develops a blue-black discoloration. If the patient survives, the demarcation and sloughing begins in one to two weeks.

Pulmonary, central nervous system, renal, and hepatic dysfunction are common; the exact cause of dysfunction is not known, but approximately 20 per

cent of patients have hemorrhage and thrombosis in the viscera.⁴

Etiology and Pathogenesis

The proposed etiologies for this disease are immune-oriented. Either a localized Schwartzman's phenomena or an Arthus's reaction have been postulated. The Schwartzman phenomena is sensitization of skin capillary endothelium by infection or toxin combined with a non-specific secondary trigger which causes IgG or IgM to attack endothelial cells. Arthus's reaction is also mediated by prior exposure, development of specific antibodies, re-exposure and subsequent antigen-antibody complex formation which leads to precipitation of these complexes on endothelium of skin vessels. Complement and neutrophils join in the injury to the endothelium.

Endothelial damage leads to release of at least two substances — Hageman factor and prostanoids. Hageman factor and previously mentioned complement, through the coagulation scheme, lead to defibrinization of plasma and local fibrin deposition while prostaglandins and thromboxanes lead to propagation of vasospasm and platelet aggregation.

Capillaries and venules are occluded, and congestive ischemia with extravasation occurs. Loss of blood volume is marked, and compensatory peripheral arteriolar vasoconstriction occurs causing more ischemia.

Biopsy specimens reveal endothelial ballooning, especially in the small venules and capillaries with fibrin-platelet aggregates occluding the already narrowed lumen.⁴ Subcutaneous extravasation with perivascular polymorphonuclear infiltrates are noted. This infiltration is speculated to be secondary to the release of neutrophil migration factor (a leukotriene) from endothelium and from tissue macrophages after the extravasation. Continued inflammatory damage leads to mural disintegration and breakdown of capillary integrity.

Treatment

To arrive at a treatment protocol, we must realize that this disease is a result of a primary illness, even though it may be inactive or subtle. Investigation with cultures, titers, and rickettsial studies may be helpful. Broad spectrum antibiotics should be initiated early to clear infection or any persistent antigen stimulation.

Helpful laboratory parameters are a CBC; a platelet count; a protime partial thromboplastin time, fibrinogen and fibrin split products; and a cortisol level determination.

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Health Problems of Southeast Asian Refugees

Anne D. Walling, M.D.,* *Wichita*

APPROXIMATELY 10,000 Southeast Asian refugees now live in Kansas. Many have moved from their original resettlement locations in quest of employment or to join relatives, but it is believed that as many as 5,000 currently live in Wichita with substantial numbers in other locations such as Kansas City, Dodge City, Great Bend, Liberal, and Salina.¹ Physicians in Kansas can therefore expect to encounter these patients from a completely different environment, particularly in primary care situations. Experiences from centers that treat numerous refugees can promote insight into the prevalent health problems in these patients and provide background information for physicians who treat them.

These refugees share the traumatic experiences of war and relocation, but the five major ethnic groups from Indochina represented in Kansas share few other common features. Not only are Vietnamese, Laotian, Cambodian, Hmong, and Chinese people culturally distinct, but some groups — especially the Khmer (Cambodian) and Vietnamese — have a long history of animosity and are likely to resent being regarded as one homogeneous population. The situation is further complicated by sub-groups within the five major ethnic groups. In addition, the previous lifestyles of the refugees cover a complete spectrum from peasant farmers to those accustomed to a complex urban environment with substantial western influence.

Health Problems

The diversity of the refugee population is reflected in studies of their health problems. Reports now are available from several centers,²⁻⁵ but each must be evaluated in the light of the ethnic group involved and the health problems assessed. As all medical students are taught, "You can't diagnose something for which you don't look." The following are among the most prevalent health problems identified in the literature.

Dental problems are reported to be present in more than 75 per cent of adults. Poor dentition and dental caries are so common as to be taken for granted which implies that refugees may not appreciate either the scope of dental services avail-

able or the value placed on good dental health in this country. To change this drastic situation would require lifestyle and dietary changes on the part of the refugees as well as massive commitments of health care money for dentistry — all in a cause that may not be perceived by the people involved as important or by health care providers as a "high priority" area.

Positive tuberculin skin test is reported in between 30 and 70 per cent of refugees depending on age and ethnic group. There is a clear gradient with age³ (*Table I*). Ethnicity may reflect previous lifestyle rather than an inherent predisposition to tuberculosis, but there are significant differences between the main groups (*Table II*) with the lowest rates reported from more rural people. It has been suggested that the length of stay in refugee camps increases the prevalence of positive skin tests.³ All adult refugees are screened overseas by chest x-ray. In all but two locations, children over two years are also x-rayed. Camps in Indonesia and Singapore x-ray children under 15 years only if related to an adult with an abnormal x-ray.⁶ Many positive tests result from inactive lesions or previous B.C.G. inoculation; however, with age and sex specific incidence rates for tuberculosis between 30 and 200 times higher among refugees than the resident United States population,⁷ there is still need for a high index of suspicion. Preventive drug therapy with Isoniazid has been widely used (*e.g.*, in 18% of all Indochinese refugees entering the United States in 1980) and is credited with contributing to the lack of transmission of tuberculosis to the American population.⁷ Those refugees who are culture positive for tuberculosis have high rates of resistance to Isoniazid (25%) and/or streptomycin (22%) but relatively low resistance (3%) to ethambutol or rifampin.⁸

Intestinal parasites. The overall rate of infection with parasites is very high (>60%) at the time of

TABLE I
POSITIVE MANTOUX SKIN TEST BY AGE

0-4	23%
5-18	44%
19-54	72%
55 +	69%

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TABLE II
POSITIVE MANTOUX SKIN TEST BY
ETHNIC GROUP

Vietnamese	69%
Cambodian	57%
Laotian	48%
Hmong	15%
	(p<.001)

resettlement.^{3, 4} Rates are highest among children (80%) and among rural people, especially the Hmong.³ Multiple infestations are reported to be very common (about 1/3 of patients) with up to ten parasites in one patient.⁹ A wide variety of parasites are reported, some of which are endemic in the United States — such as giardia, *Ancylostoma*, or ascaris — but others such as schistosoma, *Opisthorchis*, or *Paragonimus* are unfamiliar to physicians and laboratory personnel. This results in diagnostic dilemmas such as the 11 cases of the lung fluke *Paragonimus westernmani* imitating tuberculosis reported in Minnesota.¹⁰ There are hazards too for the refugees encountering endemic American parasites. At least two outbreaks of trichinosis involving a total of 15 families have been traced to refugees who were unfamiliar with the necessity to thoroughly cook pork.¹¹

Anemia is strongly related to infestation with intestinal parasites, particularly hookworm (*Ancylostoma*). Other etiologies include chronic disease (especially tuberculosis), nutritional deficiencies, malaria, or hemoglobin disorders. The overall prevalence of anemia reported varies from 16 to 37 per cent. Abnormalities of hemoglobin are common in Southeast Asia but there is wide variation with ethnic group, e.g., HBE is reported in 20 per cent of Thais and 35 per cent of Khmer. Hemoglobin A₂ is also frequent, as are thalassemia syndromes.²⁻⁴

Hepatitis B surface antigen can be detected in approximately 13-20 per cent of Indochinese refugees²⁻⁴ which is 50 times the prevalence of antigenemia in the resident United States population (0.3%).¹² They therefore represent a significant pool of infection for hepatitis B. Transmission is particularly likely to dental personnel (because of the high prevalence of dental disorders among refugees) but can occur in any surgical procedure.¹³ As refugees are primarily a young population, this is particularly significant in obstetrics. Neonatal infection with hepatitis has been reported as occurring commonly in Asian populations.¹² Long term, the role of chronic hepatitis B infection in hepatic failure or hepatocellular carcinoma has yet to be elucidated.

Venereal disease. All refugees have VDRL testing as part of a pre-visa medical screening and < 12 per cent are positive.^{2, 5} In addition to syphilis, a positive VDRL test may result from tuberculosis, malaria, or hepatitis. Other treponematoses — especially *T. pertenue* (yaws) — are common in Southeast Asia, particularly Cambodia, and may be indistinguishable serologically from syphilis. Indeed, yaws patients achieve very high titres and may never revert to being seronegative.³ Very strongly positive VDRL, particularly in a younger patient, suggests *T. pertenue*. Penicillin is the treatment of choice for all treponematoses.

Other venereal diseases — in particular gonorrhea — have been reported in refugee camps. Penicillin-resistant gonorrhea has been a particular problem in boat people as a result of rape by pirates en route to Thailand.¹⁴

Malaria is endemic in Southeast Asia, but the prevalence of cases found has been less than 4 per cent.²⁻⁵ Cases are undoubtedly being missed even on blood examination. Nationally in 1980, seven babies were diagnosed with congenital malaria. Two of these mothers had blood smears that were reported as negative but retrospectively were found to contain parasites.¹⁵ Therapy for malaria is complicated by widespread chloroquine resistance in cases due to *P. falciparum* in Southeast Asia. Complications also arise using primaquine in patients with glucose-6-phosphate-dehydrogenase deficiency (10-12% of refugees).¹⁰

Psychiatric problems are difficult to assess because of cultural differences but the trauma of previous experiences plus the continuing pressures of adjustment to an alien environment must be expected to result in a high rate of psychiatric disorder. Diagnosis and treatment of such disorders pose real challenges for psychiatric services and the refugee communities.

Other problems are reported inconsistently depending on the interest of the investigator. Hearing disorders, skin lesions, and thyroid abnormalities have been described as highly prevalent. In addition to the health problems they bring with them, refugees may fare badly with common American health problems. Respiratory infections have a reputation for being particularly common and severe in young males. Road traffic accidents and other conditions common in young populations can be expected to affect refugees disproportionately compared to the American population. Finally, practices that were normal in Asia may be unsafe or regarded as deviant here. As an example, folk medicine involving cup-

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Propranolol for Episodic Dyscontrol Syndrome

NEIL E. ROACH, M.D.*; M. DON GEORGE, M.D.† and
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PROPRANOLOL was used to treat episodic violent behavior in a patient without apparent organic brain disease or associated psychiatric disorder. The resultant family dysfunction which occurred following cessation of his violence is also examined.

The episodic dyscontrol syndrome, a poorly defined clinical entity characterized by excessive displays of anger with little or no provocation, has been addressed in American and British psychiatric literature since 1970.¹⁻⁴ The etiology of the syndrome is unknown. Maletzky reviewed the original description of episodic dyscontrol published by Mark and Ervin in 1970, and further attempted to define the syndrome.¹ Reluctant to attribute episodic violent outbursts solely to organic causes, he postulated the likely influence of psychosocial factors in the genesis of the syndrome. This set the stage for much of the work that has followed. Elliott compared neurological findings in adult minimal brain dysfunction and episodic dyscontrol in a retrospective study of 286 patients who demonstrated recurrent outbursts of uncontrollable rage.² Ninety-four per cent of his patients had evidence of organic disorders of the brain, with 119 diagnosed as having minimal brain dysfunction, in what he described as a cohort that may have been influenced by referral bias. The paper is concluded by a statement regarding the nature of violence as a learned response, recalling that twin studies have been equivocal at best in separating the effects of environmental and genetic influence upon learned behavior.

Mungas attempted an empirical analysis of the relationship of violence to causal factors, in which groups of outpatients were identified whose violent behavior differed significantly both in terms of frequency and severity.³ A specific etiology for violence was unsupported. Mungas concluded that the behavior likely resulted from multiple factors both biological and psychosocial. In a British study, Leicester supported the organicity of episodic dyscontrol, choosing to label as "temper tantrums" all episodic explosive behavior unrelated to underlying organic brain dysfunction.⁴ The validity of episodic

dyscontrol as a distinct entity was questioned, except as a descriptive term or personality trait.

While the etiology of violent behavior — specifically episodic dyscontrol — has remained unsettled, psychiatry has moved ahead in identification of new treatment methods for control of violent outbursts. One such innovation has been the discovery of propranolol for use in this setting.⁵⁻¹² Propranolol has been found useful in controlling violent outbursts following acute brain damage.⁵⁻⁶ Yudofsky *et al.* have found propranolol effective in the control of violence in children, adolescents, and adult patients with chronic brain dysfunction.⁷⁻⁸ Ratey *et al.* used propranolol to treat episodic violent outbursts in three patients with major mental disorders, with and without brain damage.⁹ Schrier found it an effective treatment for belligerent behavior associated with postencephalitic psychosis in a 12-year-old boy.¹² There have been no previous reports of effectiveness of propranolol in the control of violent outbursts in a patient with no organic brain disease and no associated psychotic disorder.

The necessity of recognizing reactions by spouse and family to the successfully treated patient, regardless of presenting complaint or method of therapy, has been documented.¹³⁻¹⁵ Kohl described previous work in this area, and identified patients who, in the course of apparently successful therapy, suffered impairment of spouse and family relationships and even recurrence of symptoms, when marital partners experienced difficulty in adjusting to improvement in the patients' conditions.¹³ Moreover, spouses and family members may begin to demonstrate pathological symptoms themselves in their inadequate attempts to redefine their relationships with the "cured" patient, a phenomenon that has been documented in psychological research.¹⁴⁻¹⁵

The following case demonstrates the possible effectiveness of propranolol on episodic dyscontrol, with special consideration given to the reaction of the wife to the patient's improved condition.

Case Report

A 32 year-old male, with no prior psychiatric history, had experienced sudden outbursts of uncontrollable anger and rage since adolescence. These outbursts had been increasing in frequency and

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severity during the several months prior to evaluation. The outbursts were usually precipitated by minimal provocation. Primary targets were inanimate objects including walls and doors. He admitted to occasionally striking individuals, although he denied having ever caused serious bodily injury. In an attempt to learn to control his anger, he had involved himself in various types of counseling for several years. Whereas he had sometimes been able to control the violent behavior by leaving the situation, the outbursts were rapidly becoming less manageable for him. He expressed concern that his behavior might threaten his present marriage.

Past medical history was positive only for gastritis. He admitted to prior heavy use of alcohol, stating that current usage was moderate. He denied use of other drugs. There was no history of head trauma, and no history of seizure disorder. Family history was positive for alcohol abuse by his father, who was also known to react violently upon minimal provocation. His mother was a stable figure in the family. Social history included a previous marriage that had ended in divorce. A high school graduate, he had maintained a good employment record despite his behavior pattern. Physical examination was remarkable only in that he was slightly overweight. Mental status examination was remarkable for unkempt appearance, anxious state, and irritable nature.

Laboratory evaluation included fasting glucose, serum electrolytes, liver enzyme profile, thyroid profile and urine analysis, and all were normal. EEG was interpreted as normal, with normal responses to photic stimulation and three minutes of hyperventilation. Formal psychological testing was not done, although he was judged of average intelligence based upon history and observation.

Lithium carbonate therapy was initiated, along with weekly individual counseling sessions, which focused upon management of his anger and his marital relationship. Adequate blood lithium levels were maintained with an oral dose of lithium carbonate of 900 mg/day. At six weeks, doxepin hydrochloride, 50 mg at bedtime was added to control nausea. This was later increased to 100 mg at bedtime. By the eighth week, the patient experienced diarrhea, ataxia, and tremor. Although blood levels did not reach the toxic range, these symptoms were attributed to lithium, which was then discontinued.

General assessment at eight weeks revealed that the patient's violent episodes were unchanged. He was started on propranolol, 10 mg b.i.d. Within one week, he noted a decrease in irritability and felt more in control of his temper. He also experienced seda-

tion at rest, but not with activity. The doxepin was gradually decreased and discontinued during the following week.

At the next visit, one week later, the patient announced that he had stopped taking all medications because of persistent sedation that caused problems for him at work. He had experienced a major loss of control four days prior to the visit. He was dysphoric, and felt the situation was hopeless. After assurance that the sedation was likely a temporary residual effect of the doxepin, and not due to the propranolol, he agreed to resume propranolol at an increased dose of 20 mg b.i.d. One week later, it was noted that he had cut his hair, had shaved, and had bathed. Personal habits had been neglected in the months prior to therapy. He related that he felt better about himself, and that his home situation had been much more calm. He continued to do well, experiencing less anger, and only occasionally needed to "walk away" from provocation. His propranolol dose was titrated to 60 mg/day without complaint of side effects, other than cold hands.

After five months, his condition remained stable. Although he continued to feel anger, he remained in control of his behavior. However, his marital relationship had deteriorated markedly during this interval. He found himself involved in an intense power struggle with his wife over parenting control and basic marital rights and privileges. He became frustrated with the situation at home, and again stopped his propranolol. His behavior deteriorated to pre-medication form. Intense supportive therapy, and initiation of his wife into individual counseling resulted in his agreement to restart propranolol at 60 mg/day, in divided doses. He continues to manage his temper without behavioral dyscontrol, despite his difficult marital relationship. Conjoint counseling with his wife has proved unsuccessful to date.

Discussion

Although little can be determined with certainty from the treatment of a single individual, it is the authors' impression that propranolol has been helpful in causing the cessation of violent outbursts of anger in this case. This conclusion is supported by the return of the symptoms when the patient stopped taking propranolol on two occasions, as well as the continued absence of symptoms while taking propranolol, in spite of worsening marital conflict. Psychotherapy and marital counseling made no apparent difference in the frequency of outbursts.

The effective dose of propranolol in this patient was 60 mg/day. Dosages have been reported in other
(Continued on page 247)

The President's Message

The Kansas Delegation to the AMA — Of People, Their Challenges and Achievements

We can be proud of the Kansas delegation to the AMA. It was my privilege to attend the AMA House of Delegates Meeting in Chicago June 17-21, and see how well we are represented by our AMA delegates and alternate delegates.

Dr. Alex Scott, a family practitioner in Junction City, is the senior AMA delegate from Kansas. He has served on a reference committee at three national meetings. He is involved in Work Group 6 of the Health Policy Agenda for the American People which deals with concern for long term funding for medical services. Dr. Scott's alternate, Dr. Warren Meyer, a general surgeon in Wichita, has been active in the Hospital Staff Section of the AMA.

Dr. Kermit Wedel of Minneapolis, another AMA delegate, is one of only two family practitioners on the 16-member Physicians Advisory Committee to the Health Care Financing Administration. Dr. Wedel's alternate delegate, Dr. Linda Warren, is a family practitioner in Hanover. Since 1981, she has been one of five women physicians on the AMA *Ad Hoc* Committee for Women Physicians in Organized Medicine. This group accomplished a great deal in completing the tasks requested by the AMA Board of Trustees, and they presented a comprehensive report at the June AMA meeting.

Dr. Lew Purinton, specialist in internal medicine in Wichita, is in his first term as AMA delegate. His alternate, Dr. Jimmie Gleason of Topeka, obstetrician and gynecologist, spoke at the AMA leadership conference in February about the Kansas Blue Shield CAP program and DRG methodology.

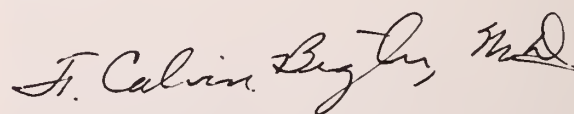
Dr. Bill Reals, pathologist and dean of the University of Kansas School of Medicine-Wichita, is AMA specialty society delegate from the College of American Pathologists, and sits with the Kansas delegation on the floor of the House of Delegates. Dr. Reals is the first Kansan elected to an AMA council, serving on the Council on Medical Education. Among other activities, he is the Council representative to the Accreditation Council for Graduate Medical Education and is in Work Group 5 of the Health Policy Agenda for the American People

which is concerned with health resources (manpower, facilities, and technology).

The strength and effectiveness of the delegation from Kansas are far greater than the small number would imply. Resolutions introduced and opinions given by the Kansas delegation were well presented. Many hours each day were spent by these delegates and several Kansas physicians who came on their own to attend caucuses, reference committee meetings, and the House of Delegates. Incidentally, on opening day, 96.3 per cent of the total delegates were present. On the three days when resolutions were considered, 354 of 355 delegates (99.7%), were seated in the House.

One example of the rapid and efficient action of the AMA was in the field of professional liability. Although several departments in the AMA deal with different aspects of the liability problem, a resolution introduced by the American College of Obstetricians and Gynecologists requested the establishment of a Department of Professional Liability. This resolution was strongly supported by the Kansas delegation and several spoke in favor of it. The resolution was acted upon and referred to the AMA Board of Trustees "with high priority." Just 48 hours later, a report from the Board of Trustees was presented to the House of Delegates announcing the creation of a Special Task Force on Professional Liability and Insurance. The composition of the task force and frequency of meetings and reports indicate that this activity will indeed have the highest priority at the national level.

The purpose of this month's "President's Message" is to introduce your representatives to the AMA. Most of you already know them. They are working hard for every physician in Kansas. We *must* work together on the challenges facing medicine — at all levels, local, state, and national. It is a struggle to meet these challenges today. This struggle demands personal sacrifice, teamwork, and just plain hard work. But the health of our patients is worth it!



President



Doin' What Comes Unnatcherly

This is being written during the world's quadrennial sports orgy when emotions and national pride are running high. Moreover, it comes to you from the gold medalist in the indolence marathon which qualifies us to comment on the matter of drug abuse in the sports world, not, as one might think first off, the well-publicized transgressions of prominent athletes but the increasing progression toward drugs to enhance physical performance and, of these, the anabolic steroids whose proven effectiveness has stimulated increasing use.

The problems of illicit drugs and those of androgens are, of course, quite different but there are two common denominators, the users and physicians. In the case of illegitimate drugs, most would agree, the physician's role lies in producing rational warnings of effect, working toward proper controls, and managing the effects when the warnings are ignored. The purpose of the steroids is to do what they are supposed to do but more than the individual is naturally capable of: build body substance and thereby improve physical (read athletic) performance, both of which have been shown to be predictable results.

To those intensely dedicated to body building and achieving the spiritual and material rewards of record-setting performance, this is all that need be said. Obviously, steroid use in prepubertal children should be excluded but the zeal of some parents and trainers warrants this mention. Women may show varying degrees of masculinization, and menstrual dysfunctions are common (but so are they in the excessive training programs such as those of long distance runners). All of the known complications of steroid use are cited as areas of risk but the proponents point to the fact that information has been gained from their use in abnormal conditions — endocrine, hematologic, oncologic. The effect on the healthy — in fact, the more than healthy —

cannot be equated with orthodox medical indications.

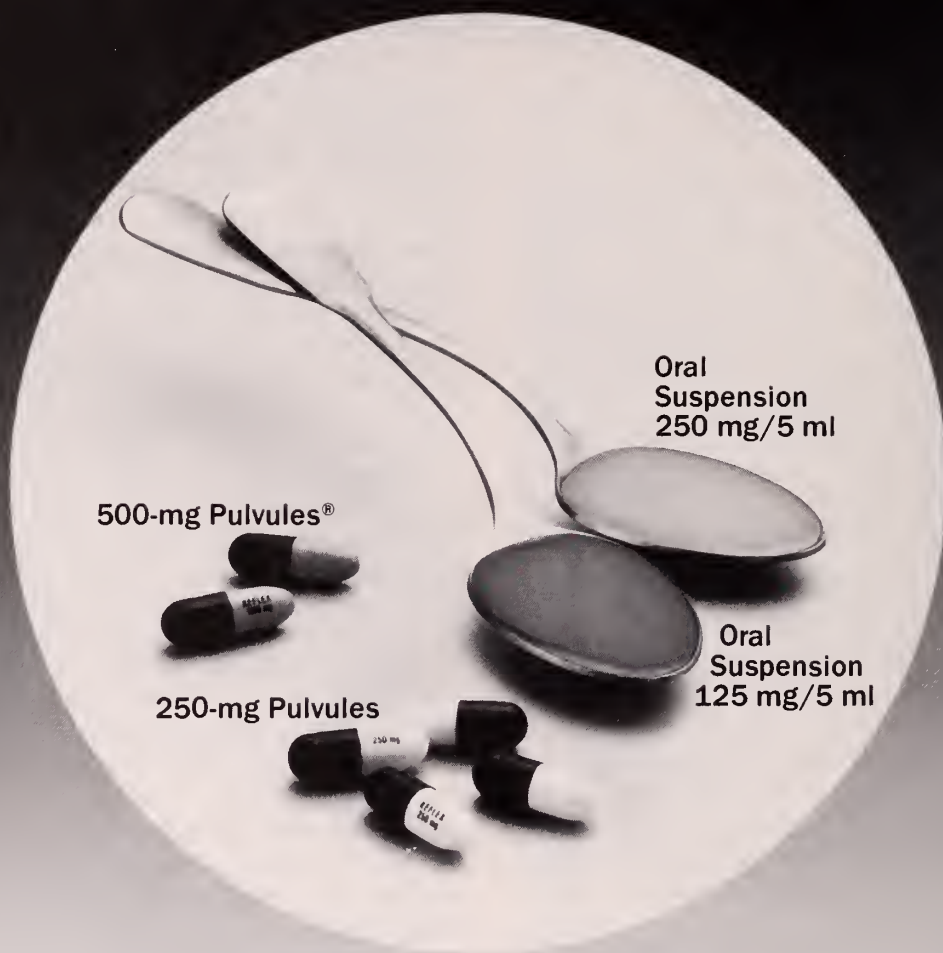
But we are not talking only about the celebrated human desire to achieve the glories of winning. The fact is that we are talking about money. Thanks to instant world communication, especially television, high athletic accomplishment is now usually measured in terms of financial benefits, not only for the athletes but the numerous agencies along the way that will profit. We obviously then are not talking about the true amateurs who are pursuing their various activities for personal satisfaction and in relative privacy — unlikely prospects for reliance on drugs, licit or illicit, since they are free of the pressures to which drug use is usually attributed. But those who qualify as the highest achievers and have the greatest impact on the public, especially the young public, may well influence the athletic mores and standards that will serve as goals for those coming on and invite achievement not only by personal effort but by their ability to devise effective artificial conditions.

The winning of an athletic contest by an individual is presumed to indicate that that individual has, at the moment, the best physical and mental capability among the contenders. But if that capability is due, in addition, to the introduction of agents of extrinsic origin, however natural their basic character, shouldn't the laboratory rate at least a footnote of appreciation? Shouldn't the record books at least put an asterisk by the performance? If chemical and biological artifices are acceptable, what of mechanical?

Given the tacit legitimacy of steroid use in various medical conditions, the physician becomes the logical source for their procurement. Is it the physician's proper role to provide substances of such demonstrated potency when the purpose is not the alleviation of disease or even the avoidance of disease but the improvement of athletic performance beyond the person's innate ability? It will be argued that the performance benefits are so well known to athletes and their trainers (out in the "real world," as it has been put) that, failing procurement from legitimate sources, they will get them one way or another — the common argument when control of an agent of questionable legitimacy or potential risk is sought.

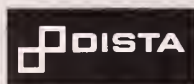
It hardly seems an appropriate medical function but it will be interesting to see how the proponents work around to make it seem legitimate. After all, it's too important — like money. — *D.E.G.*

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SE Asian Refugees

(Continued from page 239)

ping can leave marks on a sick child resulting in charges of child abuse. Similarly, a group of Laotians were poisoned by wild mushrooms¹⁶ in spite of applying what would have been in Laos a reliable test for toxicity. In Laos, all poisonous mushrooms turn red when boiled with rice. This is not true for all American poisonous mushrooms; hence seven people became ill even after testing the fungi.

Summary

In summary, the Southeast Asian community is a heterogeneous group that presents both exotic and more mundane health problems for diagnosis and treatment. Complicating the expanded differential diagnoses are major problems of communication and cultural embarrassment. The general comments prepared by the Center for Disease Control in 1979 have been borne out in practice, *i.e.*,

- The majority of refugees will be free of major contagious disease.
- Where an illness is present, it will likely represent a personal rather than a public health problem.
- The main health problems, perhaps exceeded only by the stress of resettlement itself, include tuberculosis and parasitic disease.

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Purpura Fulminans

(Continued from page 237)

Treatment should initially be supportive with generous fluids and metabolic correction. Aggressive management would include monitoring with either a pulmonary artery or central venous pressure catheter, an arterial line, and a Foley catheter. Fluids should be pushed to just short of fluid overload to maintain an adequate tissue perfusion and adequate urine output. This is necessary because of the marked volume depletion ("third spacing") from capillary leakage. Avoidance of vasoconstrictors for blood pressure support is suggested in every therapeutic review.

Because of their ability to block antigen-antibody complex formation, decrease prostaglandin synthesis, inhibit neutrophil migration, enhance capillary stability, and support cardiovascular function, glucocorticoids have been tried. One speculated disadvantage is that steroids enhance the Schwartzman phenomena in laboratory animal models. However, heparin has been shown to block the cortisol-induced generalized Schwartzman reaction.⁸

To approach the vaso-occlusive phenomena, some have suggested streptokinase, which acts with plasminogen to produce plasmin, a fibrinolytic substance which degrades clots. Others have blocked fibrinolysis with epsilonaminocaproic acid to decrease fibrin split products, which inhibit coagulation systemically; however, the results have been variable.⁹

It would seem that heparin with its local action on the endothelium to stabilize membranes, prevent coagulation cascade activation, and stimulate antithrombin III activity to prevent further fibrinolysis would be an optimal drug for the local effects on purpura fulminans (recommended dose 100 units/kg/4 hrs intravenously). Vitamin K and

FFP would be helpful after heparinization to provide factors that were depleted locally, maintaining the circulating coagulation system for other organs.

Success has been shown with hyperbaric oxygen therapy for treatment of the ischemic regions.¹⁰ However, this modality has limited access.

Summary

The literature recommends early institution of heparin and broad spectrum antibiotics as volume depletion is being aggressively treated, followed by corticosteroids and coagulation factor replacement after the patient is heparinized. Close monitoring of the cardiac, respiratory, renal and hematologic systems is necessary.

Survival is less than 50 per cent overall, but early debridement, aseptic wound care, and functional splinting for extremities is essential for an optimal outcome.

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Propranolol

(Continued from page 241)

studies to range between 60-1500 mg/day,⁸⁻⁹ with the elderly possibly requiring lower dosages.¹⁰

The side effects of propranolol most commonly noted — hypotension and bradycardia — were not observed in this patient. When reported in previous studies, they usually have been well tolerated by patients.⁷⁻¹² It is conceivable that the patient's report of cold hands could have been related to the propranolol; however this complaint occurred during bitter cold winter weather.

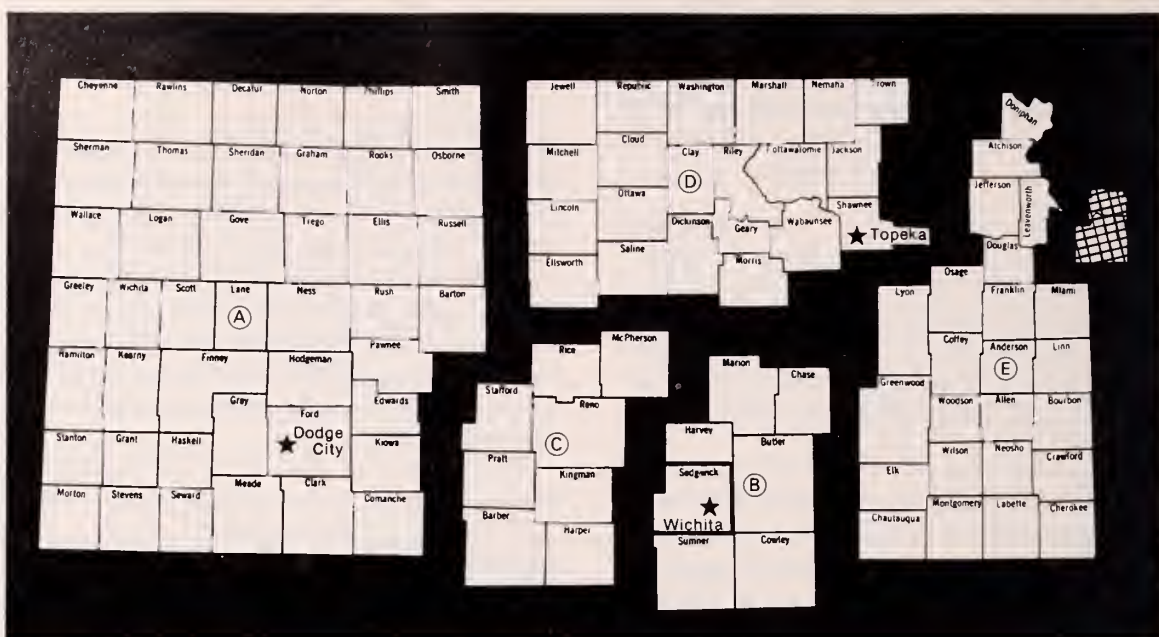
The mechanism of action of propranolol in controlling violent behavior is incompletely understood. Williams *et al.* summarized previous speculation regarding mechanism of action.⁸ They proposed that its effect might be due to CNS membrane stabilization, or perhaps an effect upon cerebral catecholamines. While this case provides no additional information regarding mechanism of action, it does render questionable speculation that propranolol acts by correcting some defective chemical balance altered by identifiable organic brain disease. Clinical evaluation of this patient showed no evidence of organic brain disease.

The negative response of the patient's wife to his improved condition and the impact of this response upon the success of his treatment have been significant. As he became less afraid of violent outbursts, he began to take a more active role in the discipline of his wife's children by a previous marriage. She began to challenge his control by finding fault with his behavior, and refusing sexual intercourse frequently. Feelings of mutual incompatibility and defensiveness occurred between them. This eventually led to non-compliance with medication, followed by a return of symptoms. Similar responses by marital partners have been reported with other symptoms.¹³ This case illustrates the difficulty that can be encountered attempting to help a couple cope with such a response.

Summary

The use of propranolol in the treatment of the episodic dyscontrol syndrome shows promise for future research. At the same time, attention must be paid to changes in family relationships, which may occur following the cessation of episodes of dyscontrol, if the potential benefit of such treatment is to be fully realized.

References are available from Dr. Roach, UKSM-W, 1010 No. Kansas, Wichita KS 67214.



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Ovarian Tumors

(Continued from page 233)

The masses in the right ovary (apart from the corpus luteum) are consistent with changes described as "almost invariably" found in the contralateral ovary of patients with thecoma.³ These changes range from diffuse cortical hyperplasia to nodular masses histologically identical to thecoma. The term "diffuse thecomatosis" has been applied to this process and an association suggested with the development of many types of female cancer.

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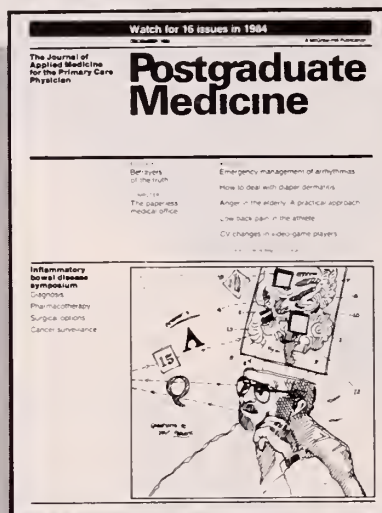


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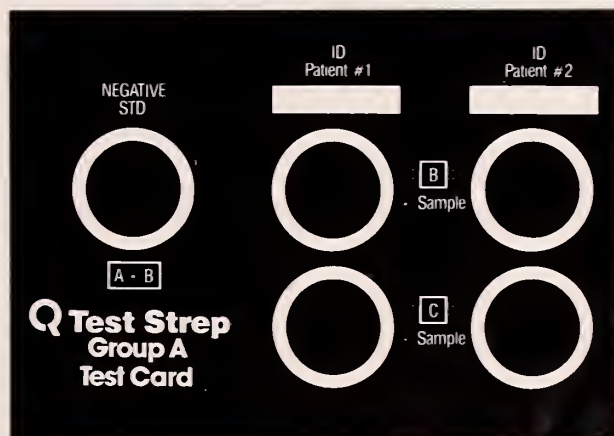
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Departmental Reports

(Continued from page 230)

needs and to develop programs to meet those needs. Future activities will include the third annual Family Practice program.

Another program teaches improved educational skills to our faculty. Our Annual Shaw Memorial Lectureship will be presented for the second time this year, and the Ernie Chaney Postgraduate Mini-seminar series will be implemented.

Research efforts have been enhanced by the addition of a Director of Research and a Head of Community Medicine. Installation of a new computer at the Medical School has enabled us to begin storing data for applied investigative research in Family Practice. A series of seminars on the development of skills in investigative studies has recently been initiated for physicians in Kansas and the immediate perimeter area.

Investigative studies are being conducted in the area of perinatology and infant and fetal mortality by members of the department in conjunction with the Wichita-Sedgwick County Health Department.

The department has made significant progress in achieving the goals and objectives developed for meeting its responsibilities at UKSM-W. The department's future activities will be directly related to state needs for family physicians, to the state economy, and to the University of Kansas administration.

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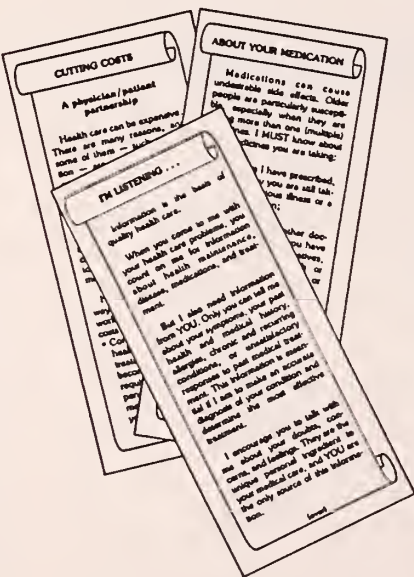
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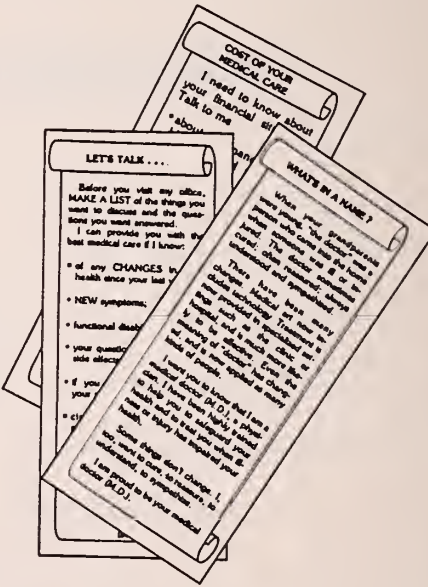
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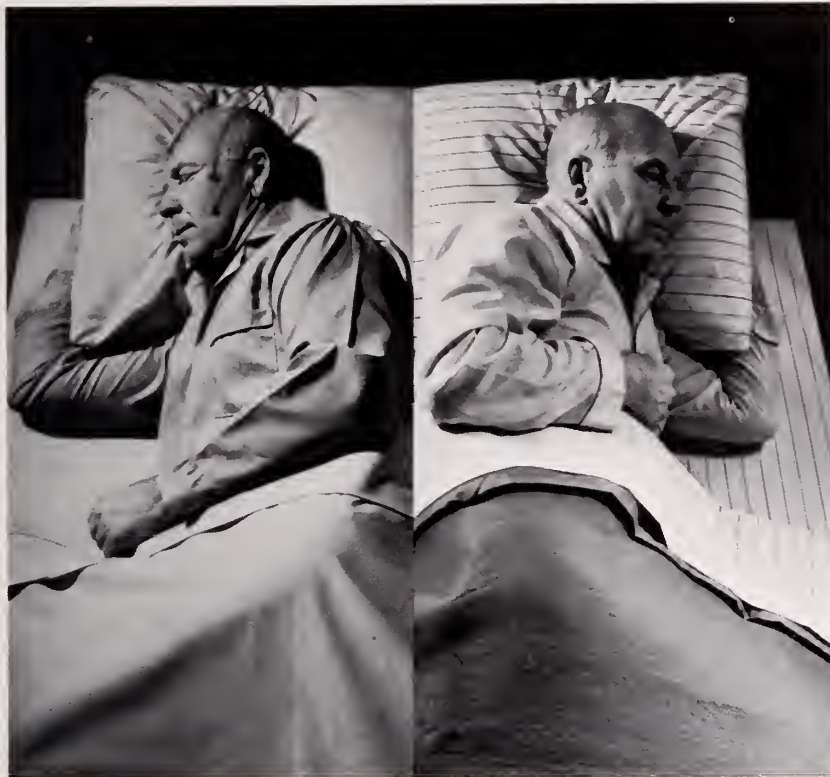
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References: 1. Kales J et al: *Clin Pharmacol Ther* 12:691-697, Jul-Aug 1971. 2. Kales A et al: *Clin Pharmacol Ther* 18:356-363, Sep 1975. 3. Kales A et al: *Clin Pharmacol Ther* 19:576-583, May 1976. 4. Kales A et al: *Clin Pharmacol Ther* 32:781-788, Dec 1982. 5. Frost JD Jr, DeLucchi MR: *J Am Geriatr Soc* 27:541-546, Dec 1979. 6. Kales A, Kales JD: *J Clin Pharmacol* 3:140-150, Apr 1983. 7. Greenblatt DJ, Allen MD, Shader RI: *Clin Pharmacol Ther* 21:355-361, Mar 1977. 8. Zimmerman AM: *Curr Ther Res* 13:18-22, Jan 1971. 9. Amrein R et al: *Drugs Exp Clin Res* 9(1):85-99, 1983. 10. Monti JM: *Methods Find Exp Clin Pharmacol* 3:303-326, May 1981. 11. Greenblatt DJ et al: *Sleep* 5(Suppl 1):S18-S27, 1982. 12. Kales A et al: *Pharmacology* 26:121-137, 1983.

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Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, light-headedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, e.g., excitement, stimulation and hyperactivity.

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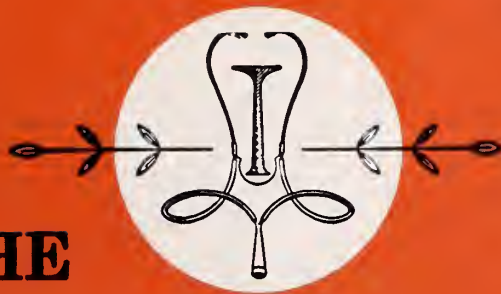
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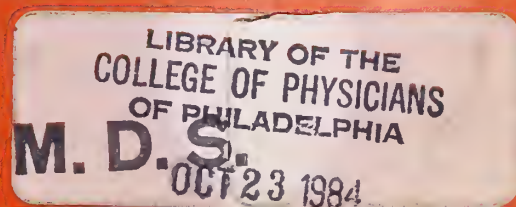
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Address all correspondence to the JOURNAL OF THE KANSAS MEDICAL SOCIETY, 1300 Topeka Avenue, Topeka, Kansas 66612; 913-235-2383; toll free in Kansas, 1-800-332-0156. Manuscripts should be submitted to the Managing Editor. Refer to "Information for Authors" for details.

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The President's Message

What Can You Do About the Malpractice Crisis?

More than \$2 billion will be spent nationally for malpractice insurance premiums in 1984. A major cost in the current litigious climate is the ordering of additional tests or treatments primarily to prevent or defend against threat of suit. Additional costs occur due to excessive record keeping and time spent directed solely at preventing malpractice claims. Surveys have estimated that the costs of positive defensive medicine make up 25-50 per cent of the total cost of medical treatment. The cost and medical risk of withholding a helpful diagnostic test or a possibly beneficial procedure because of fear of suit cannot be estimated. These are only a part of the staggering societal costs of professional liability.

There is national political preoccupation with cutting the costs of Medicare. The media give a great deal of attention to the high costs of health care. Is it not surprising — and shocking — that there is so little attention given to such a major factor in the health care cost picture? Why has there been so little notice of a phenomenon that is straining — and may wreck — our generally excellent health care cost picture?

Malpractice premiums are expensive to the physician, but the public eventually pays the cost. Even though we physicians have frozen our fees by voluntary restraint, the cost of skyrocketing premiums will be shifted to the patient. Less time spent per patient visit is a logical consequence of increasing overhead. This promotes a vicious cycle. The tighter the cost-time squeeze, the less opportunity to establish and nurture a sound physician-patient relationship which is the best preventive measure against litigation.

Economic considerations of an issue are important and are needed in any rational discussion. However, there are more important considerations than money. To think in terms of money alone, we would be trapped into the same fallacy inherent in malpractice law — namely, that all wrongs can be made right with the application of a green-back poultice.

Francis W. Peabody, M.D., delivered an address to the students at Harvard Medical School entitled: "The Care of the Patient." That lecture was published in the *JAMA* in 1927 and has been reprinted as a Landmark Article in their August 10, 1984, issue. This article should be read by every practicing physician. Dr. Peabody's address ends with the statement: "The secret of the care of the patient is in caring for the patient."

As we care for our patients, we as physicians must stop the development of an adversarial confrontation. What has been traditionally a physician-patient relationship must not be allowed to evolve into a defendant-accuser situation. As each year goes by, I find that I more frequently ask myself as I start interviewing an unknown

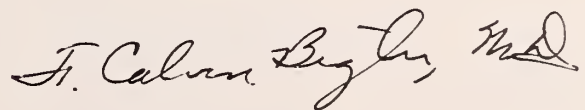
patient in the middle of the night in the emergency room: "Is he (or she) going to sue me?" This is not conducive to the practice of good medicine.

The public perception of the malpractice picture is itself part of the problem. The patient often perceives that an inordinately large sum of money is available to compensate for a bad result of treatment, even when nothing may have been done improperly. One hears from the public such statements as: "There's a big pile of money in Topeka and I want to get part of it." Once locked into the legal process, the patient is imprisoned often for years in an atmosphere wherein his or her life is dominated by a single fixation — monetary vindictiveness toward a doctor who was initially dedicated to caring for that patient. This situation frequently harms the quality of life of that person and produces a protracted tragedy. That patient — or any other person — may approach his (or her) first contact with another physician with the attitude: "Is this doctor suable?"

We as Kansas physicians have a great challenge. What can we do about the malpractice crisis? First, we must ourselves perceive the problem(s). Next, we must enlighten the citizens of Kansas. And finally, we must legislate reforms, since professional liability is governed by state law.

I would recommend that each one of us be more candid with our patients and with the citizens of our community. Be prepared to discuss the malpractice problems and remedies. Let your patients know the direct cost of malpractice premiums. As an example, in my office there are two surgeons. Our malpractice premiums in 1982 accounted for 3.5 per cent of our total office overhead. In 1983 that part of the overhead was 6.7 per cent, and in 1984 the malpractice premiums make up 12.7 per cent of our office overhead. And we carry the minimum of insurance required by law!

We as physicians must perceive the threat of the present malpractice crisis and be able to relate it to the perception, appreciation, and political mobilization of our patients, the public, and our state legislators. Malpractice is a threat not only to the physician. The current malpractice crisis is a threat to the health care of our patients and to every citizen in our state. We must all work together to improve the health care of Kansas. That is what you and we must do about the malpractice crisis. We all — as physicians, patients, and citizens — must eradicate this disease!



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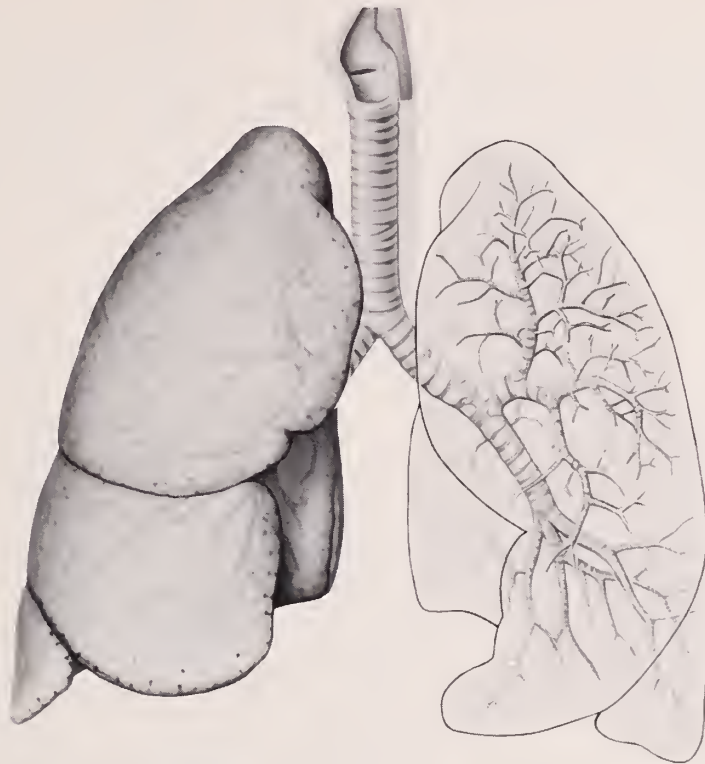
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Indications and Usage Cecilor* (cefactor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae* (*Diplococcus pneumoniae*), *Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci).

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Cecilor.

Contraindication Cecilor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings IN PENICILLIN SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS. AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Cecilor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad spectrum antibiotics including macrolides, semisynthetic penicillins, and cephalosporins; therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, manage-

ment should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out.

Precautions *General Precautions* — If an allergic reaction to Cecilor* (cefactor, Lilly) occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of Cecilor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Cecilor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended. As a result of administration of Cecilor, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinistix* tablets but not with Tes-Tape* (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy — *Pregnancy Category B* — Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum

human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Cecilor* (cefactor, Lilly). There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers — Small amounts of Cecilor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.16, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Cecilor is administered to a nursing woman.

Usage in Children — Safety and effectiveness of this product for use in infants less than one month of age have not been established.

Adverse Reactions Adverse effects considered related to therapy with Cecilor are uncommon and are listed below.

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 10). Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100), pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthritis and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Cecilor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have

occurred in patients with a history of penicillin allergy.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain — Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic — Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic — Transient fluctuations in leukocyte count predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal — Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

(061782R)

Note Cecilor* (cefactor, Lilly) is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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Contraindications: Concomitant use with other potassium-sparing agents such as spironolactone or amiloride. Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K⁺ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K⁺ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids, and during concurrent use with amphotericin B or corticosteroids or corticotropin [ACTH]). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Cumulative effects of the drug may develop in patients with impaired renal function. Thiazides should be used with caution in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease. Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic and hemolytic anemia have been reported with thiazides. Thiazides may cause manifestation of latent diabetes mellitus. The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide; dosage adjustments may be necessary. Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported. Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Anti-hypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. Triamterene has been found in renal stones in association with the other usual calculus components. Therefore, 'Dyazide' should be used with caution in patients with histories of stone formation. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide'. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased dietary intake of potassium-rich foods. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function.

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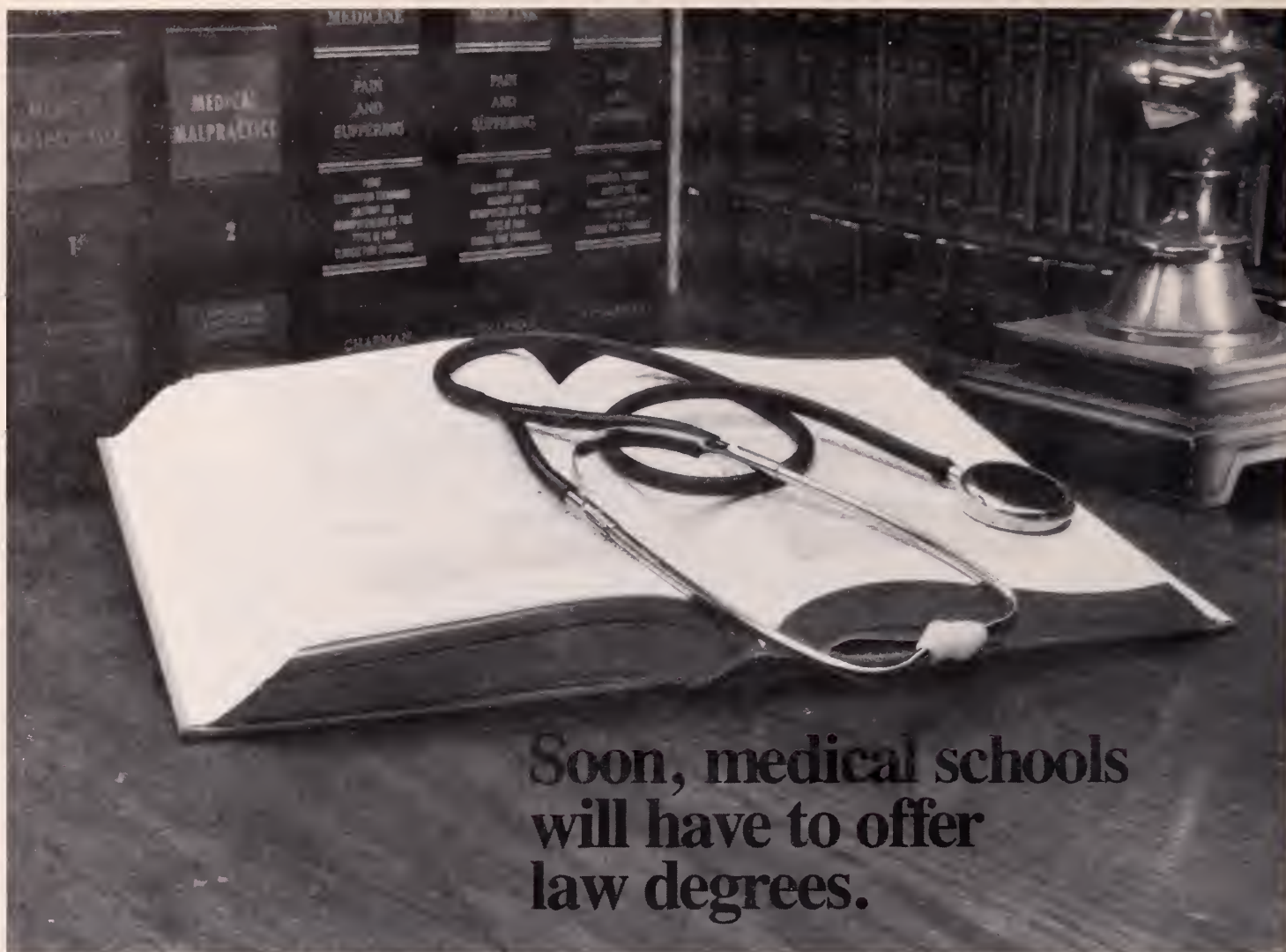
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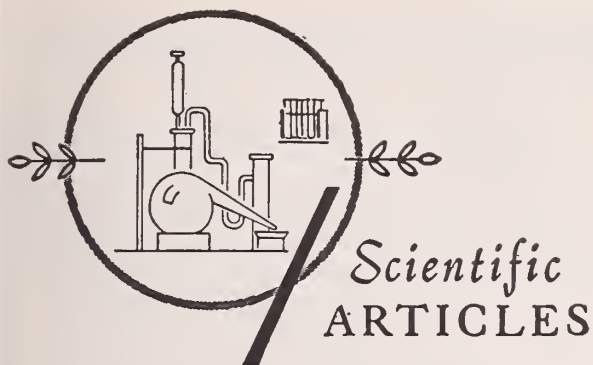
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Chronic Pain: When Aspirin Doesn't Work

EDWARD J. NOVAK, M.D.,* *Topeka*

THE SEARCH for some way to relieve pain has long provided a major impetus to the development of the medical profession. The problem of pain control occupies as much as 60-70 per cent of family practice. Pain clinics have become nationwide and are proliferating steadily. Aside from mere prevalence, pain has many serious consequences justifying the widespread concern. Since pain is one of the most potent inhibitors of activity, the classical patient who hurts is unable to carry out normal self care functions. S/he drops out of the employment market, becomes withdrawn, depressed and joins the millions going from doctor to druggist to hospital desperately looking for relief. The real cost to industry and to society as a whole is a major part of the nation's health care expense. In spite of the fact that pain has afflicted humankind since homo sapiens first evolved, the exact mechanism and physiology remains a mystery. The very existence of pain is often questioned. Aside from those cases involving individuals faking pain for whatever reason or those with psychological derangements, pain is always real to the person who is feeling it.

There is a consensus about some basic facts. It seems apparent that pain is initiated or first perceived in a receptor organ. This has been identified as a free nerve end, and these are found throughout the body. Impulses are then transmitted by means of various sized myelinated and unmyelinated fibers to the posterior horn of the spinal cord. The pathway in

the cord is located in the posterior gray column. Tracts in this column end in synapses with neurons in the posterolateral or medial ventral nucleus of the thalamus.

Sensation is processed in the reticular activating system and cortex and then produces an effect on the consciousness. This results in the person's reaction to the pain. Final presentation or appreciation is influenced by the individual's state of awareness and apprehension; by possible consequences such as gain or punishment; by intelligence; by memory of similar pain in the past; and by the intensity of the stimulus. Pain can be interrupted or ameliorated by attacking it at any point along this circuit.

Obviously, the ideal cure is to eliminate the cause, whatever it is, that stimulates the receptor. Since this is not always possible innumerable approaches have been designed to dampen the receptor organ's ability to react or to change the reaction to a more acceptable form of sensation. Simple pressure in the form of massage can be effective. The application of pharmaceuticals such as lotions, ointment, and sprays has its place. Heat and cold in countless forms are used with varying degrees of effectiveness.

The conducting nerve is accessible for interruption or modification. Cooling a nerve, by whatever means, will decrease its conduction velocity and reduce the number of painful impulses transmitted. Ultrasound will interfere with neural membrane polarization even to the point of a complete block in conduction. There are a number of drugs that can be injected into or in the vicinity of a nerve to alter conduction. Drugs such as lidocaine or procaine chemically change the electrical charge of neural membranes. This affects the capacity to transmit impulses. Other chemicals such as alcohol or phenol

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act by destroying myelin or the axon of nerve fibers. The extent and duration of the destruction depends on the strength of the chemical and the amount used. The site of injection is also a consideration. Depositing a substance in the nerve itself usually results in greater effect than infiltrating the surrounding tissue. Pathways in the spinal cord are not as easily accessible. Intrathecally injected local anesthetics and analgesics will produce temporary loss of sensation. Phenol and alcohol similarly destroy conducting neural tissue and produce a lasting block in transmission. Surgical intervention at the root or cord level can permanently disrupt sensory pathways including the ones carrying pain impulses. There are countless approaches to modify the neural activity triggered by a painful stimulus at the intracranial level. The most common and probably easiest method of affecting the brain is through the use of pharmaceutical agents. An exhaustive list of drugs used to alter pain perception and integration at the cortical level would literally make up a sizeable volume. The list would include non-narcotic analgesics, narcotics, sedatives, hypnotics, tranquilizers, and anesthetics.

The complexity of neural activity at this level seems to increase its vulnerability to a great number of techniques. Where else in the body does an inert chemical, a placebo, act to produce subjective and objective physiologic changes? In keeping with recent concepts, such an inert chemical has no measurable pharmacological effect. Apparently the expectations, the suggestions that overtly or tacitly accompany the taking of a drug cause certain tissues to produce painkilling substances named endorphins. The end result is the same whether the anticipated relief is suggested by external influences or originates in the patient's mind.

The appreciation of pain can be dulled, altered, or even blotted out by a number of medications. Unfortunately, all effective drugs have side effects that may be intolerable or even life-threatening. The search for an ideal analgesic continues. Such a drug should be absorbable when taken by mouth, rapid acting, long lasting, and free from addictive properties and toxicity.

At the cerebral level pain becomes an emotional experience and emotions can be influenced by thoughts. Distracting a person's concentration from the pain often helps ameliorate the discomfort. This cortical distraction may explain, in part, the success often seen with a program of graded exercise. The patient who has been forced into inactivity by pain is guided through a series of progressively complicated, difficult, and functional activities. The combination of successful performance and diversional

attentiveness often raises the pain threshold dramatically. In addition the exercises produce such well known benefits as improved strength, skills, cardiorespiratory function, and joint range of motion.

In the presence of anxiety and apprehension pain is subjectively intensified. Procedures to reduce anxiety may be effective in raising the pain threshold and modulating reactions to pain. Basic relaxation techniques such as Jacobsen's regimen may result in as much relief as a sedative. Profound relaxation is one of the features of a hypnotic trance, and hypnosis has proved dramatically effective in appropriate cases. Hypnosis is far more complicated than the simple induction of relaxation. It includes imagery, detachment, and regression among other phenomena but the decrease in muscle tone and tension is a strikingly obvious feature.

The element of relaxation is also encouraged in most biofeedback techniques — with the exception of procedures designed to purposefully initiate and encourage muscular activity such as in paresis.

The results of the Menninger group in alleviating headache due to migraine are widely known. However, several studies seem to indicate that biofeedback is more effective in preventing pain than in abolishing established pain.

Acupuncture is another approach to pain control. Physician reaction to acupuncture is analogous to the six blind men exploring an elephant. There are reports showing a high percentage of dramatic relief in painful conditions. There are also reports recording a lower-than-placebo effect with equivocal results. The literature is replete with statistics documenting results falling between these extremes.

One obvious reason for the marked variations in evaluating the effects of acupuncture is the fact that it is no longer a single technique even in the Orient. Traditional procedure involved the placement of silver needles and gold needles in rigidly delineated points on meridians — designated lines traversing the body. Today any kind of needle seems to be acceptable. One group of operators inserts a needle without further manipulation. Another insists that constant movement of the needle is essential. Yet another advocates the use of a low frequency current delivered through the needle.

There are also practitioners who have substituted pressure for the needling. The pressure may be exerted manually or with the aid of instruments. There is no longer any agreement on the site of puncture, although one approach has simplified this problem by developing "auriculo-therapy." All body parts are allegedly represented on the external auricle and

(Continued on page 279)

Diagnosis and Management of Endometriosis

Jerome S. Menaker, M.D., *Wichita*

ENDOMETRIOSIS, although first reported by Rokitski in 1860, received little attention until Sampson's reports in 1921.¹ His theory of retrograde menstruation as a cause stimulated much clinical and experimental activity. In addition, Robert Myer's studies on anaplastic and metaplastic changes help account for cases not explainable by menstrual regurgitation. More recently, laparoscopy has enhanced both the study and treatment of the condition.

The resistance of some women to the condition, its failure to occur consistently in the presence of menstrual obstructions, evidence of familial predisposition, and reduced incidence as a result of early and frequent pregnancies remain unexplained.

Pathology

Typical endometrial glands surrounded by stroma are the basic lesions with inflammatory cells, red cells, and hemosiderin apparent in larger cysts. Most frequent sites of implantation are the ovaries, cul-de-sac, uterosacral ligaments, tubes, bladder, and rectosigmoid peritoneum. Other pelvic structures occasionally are involved as well as distant sites such as laparotomy scars, breasts, lungs, and pleura. Involvement of the sciatic nerve has been recognized more recently as a troublesome focus. The size of the cysts may be a factor with rupture of the large ones producing emergencies. Adhesions, scarring, and obstruction due to associated inflammation often require surgical attention. Malignancy is rare.

Incidence

Earlier impressions (rarity of the condition before age 35 and in blacks as well as frequency in higher socioeconomic groups of Caucasians) have been revised as a result of information obtained by laparoscopy which has shown it to be frequent in the 20s (often as early as age 17 yrs)² and equally common in the black and yellow races.

Symptoms

Acquired and progressive dysmenorrhea, usually continuing a day or two after menses have ceased, is the most common symptom with dyspareunia, menstrual disturbances, dyschezia, infertility, and

chronic pain following in that order. There is, however, no reliable correlation between symptomatic pain and the extent of the disease, distressing pain sometimes being associated with minimal lesions while large endometriomas may be asymptomatic unless they rupture.

Infertility, other than that associated with tubal scarring, fibrosis, and impaired motility, is frequent but not well understood. An increase in peritoneal fluid proportional to the severity of the disease is found and increased prostaglandins have been measured and associated with altered tubal motility and rate of luteolysis. Increased peritoneal macrophages and phagocytosis of sperm are associated with endometriotic infertility.³⁻⁵

A protein antigen present in menstrual fluid and trapped in endometrial implants has prompted interest in an autoimmune response with possible rejection of early implanted embryos or interference with sperm passage.

A spontaneous abortion rate of 44-47 per cent (possibly due to high prostaglandin levels) has been found, but conservative surgery has reduced this to 8 per cent.

Treatment

Treatment methods include endocrine, surgical, and (rarely) radiation techniques. They can be classified as palliative or curative in relation to the severity and extent of the disease and interest in child-bearing, with alleviation of pain (spontaneous or coital), minimizing of abnormal bleeding, and improvement of fertility as objectives.

Early, minimal endometriosis may require only palliative analgesics, prostaglandin inhibitors, and six-month followups. Preparatory to treatment, certain precautionary measures are in order. Pelvic examination should be gentle to minimize possible forcing of endometrial tissue out through the tubes. Insufflation should be limited to the proliferative phase to avoid inadvertent interruption of possible pregnancy and to minimize inflation of viable endometrium through the tubes. Cervical obstructions which might enhance retrograde menstruation or impede sperm passage should be corrected. When endometriosis is suspected, laparoscopy is considered essential for adequate visualization, staging and possible biopsy, the two puncture technique providing the most information.

Presented at the annual meeting of the Kansas Chapter of the American College of Surgeons, September 11, 1983.

Endocrine Therapy

This should be temporary and is more effective early in the disease with pregnancy following as soon as possible. If pregnancy is not the goal, an-ovulation may be sought by estrogens, androgens, progestins, or some combination of them. Estrogens have been shown to produce regressive changes and obviate surgery.

Androgens, although effective, carry the known risks of masculinization in the necessary dosages. Pseudopregnancy can be induced by the combination oral contraceptives in mild to moderate cases if pregnancy is not an immediate aim, with the low estrogen-high progestin dosages preferred. Dosage is continuous, increasing as guided by break-through bleeding, but limited to six to nine months. Long-lasting intramuscular forms are undesirable unless pregnancy can be deferred for at least a year.

An antigonadotrophic impeded androgen, Danazol, was released for clinical use in 1975 and has probably been the most effective hormonal treatment, although temporary. It produces atrophy of the endometrial tissue and other indications of hypoenestrogenism. Conception usually follows one to four months of therapy. A therapeutic trial of two divided doses totalling 400-800 mg/day for six to nine months is advised. Weight gain, edema, menopausal-type symptoms, and some masculinizing effects are additional drawbacks. These adverse features combined with the cost of about \$150 a month pose limiting factors to its use.

In terms of achieving pregnancy, hormonal therapy can be considered reasonably effective (with some allowance for individual bias of various investigators) with approximately 50 per cent success, reports ranging from 10 to 72 per cent.

Surgical Therapy

Surgical therapy ranges from laparoscopic surgery through various degrees of conservative management to the definitive curative surgery of total hysterectomy with bilateral salpingo-oophorectomy. Since the crux of the matter is the condition's dependence on ovarian activity, the choice is determined by the extent to which eventual ovarian function is desired. Conservatism is in order since the progression of the condition is slow. Non-surgical methods are available with the promise of advances, malignant change is rare, and the eventuality of the menopause will resolve the matter.

Laparotomy should be meticulous with careful hemostasis and sharp dissection of adhesions, elimination of all apparent lesions, and scrupulous peritonization. The efficacy of pseudopregnancy prior to

surgery is undetermined. Excision of endometrial cysts, myomectomy where leiomyomas coexist (about 15% of cases), and subsequent peritoneal toilet with isotonic saline are recommended. The value of dexamethasone or promethazone in connection with surgery is unsettled.

A diagnostic problem obtains when endometriosis exists within the sciatic nerve sheath since severe pain, muscle weakness, and atrophy of the muscles may suggest an intervertebral disc herniation, osteoarthritis, intraspinal tumor and various other orthopedic or neurologic conditions, but the presence of a cyclic association with the menses gives a clue to the origin of the symptoms. At surgery, a bluish puckering over the sciatic nerve near the ureter in the floor of the pelvis may indicate the site of involvement and consequently careful dissection of the area with removal of endometrial implants is in order. If this is impossible without damage to the nerve, surgical castration will be effective. The formerly used radiation therapy has now been abandoned.

Surgical therapy approximates hormonal therapy in resulting pregnancy rates, and possible improvement can be achieved with 8-12 weeks of pseudopregnancy postoperatively. In severely symptomatic cases when pregnancy is not of concern, removal of the uterus, tubes, and ovaries remains the ultimate solution with radiation reserved for only those cases where malignancy is ruled out and surgery is contraindicated.

Summary

Endometriosis is one of the most frequent afflictions of women. High suspicion, vigorous investigation, and diligent treatment can be gratifying to both patient and physician. The laparoscope has revealed a higher frequency than previously believed, and has confirmed the prevalence of this condition in the black and yellow races as well as white. Hormonal and surgical therapies are applied in accordance with the selection of cases, duration and severity of the disease, and objective of treatment. Pregnancy rates following either course of therapy are approximately 50 per cent. Danazol, a relatively new hormone, shows promise, although only removal of the uterus, tubes, and ovaries is truly curative.

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(Continued on page 277)



Current COMMENT

H. B. LINDSLEY, M.D., *Editor*

Hemostasis: A Practical Diagnostic Approach

Barry S. Skikne, M.D.* and Marjorie L. Zucker, M.D.,† *Kansas City, Kansas*

DISORDERS of hemostasis are a frequent problem in most areas of clinical medicine. They can be classified into abnormalities of the blood vessels, platelets, or coagulation factors. Hereditary coagulation factor defects usually involve a single coagulation protein, while the acquired coagulation factor disorders, which are more common, generally involve multiple clotting factors, and often have associated platelet and vascular abnormalities.

Clinical Diagnosis

Clinical history and physical examination are important in the evaluation of patients who present with hemostatic disorders, and guide the selection of laboratory investigations. A family history of bleeding, the sex of affected family members, the age at onset of bleeding, and a history of bleeding after injury, obstetrical and surgical procedures, or tooth extraction should be sought. A careful drug history should be elicited and any underlying clinical diseases documented, such as liver disease or renal failure. Bleeding manifestations in patients with coagulation factor abnormalities characteristically differ from those in platelet or vascular disorders (*Table I*) and the type, distribution, and severity of bleeding should be carefully determined. In patients with defects confined to primary hemostasis such as thrombocytopenia, functional platelet defects and von Willebrand's disease, formation of the platelet

plug is delayed, leading to capillary, mucosal, and intra-operative bleeding. Secondary hemostasis, or platelet plug consolidation by fibrin, is however, relatively normal. On the other hand, in patients with defects of coagulation, *e.g.* hemophilia, primary hemostasis is normal, but consolidation of the platelet plug is delayed. Intra-operative hemorrhage is thus less usual, while delayed hemorrhage is commonly seen.

Laboratory Diagnosis

Initial screening tests in a patient with abnormal hemostasis will differentiate between a vascular, platelet, or coagulation disorder (*Table II*). The template bleeding time and tourniquet test are usually abnormal in vascular disorders, as well as quantitative and qualitative disorders of platelets. In vascular

TABLE I
TYPES OF BLEEDING SEEN IN DISORDERS
OF HEMOSTASIS

	Coagulation Disorders	Platelet and Vascular Disorders
Petechiae/Pupura	rare	characteristic
Mucosal bleeding	uncommon	common
Bleeding from superficial cuts	minimal	persistent
Ecchymoses	common (usually solitary)	common
Hemarthroses	common	rare
Intramuscular bleeding	common	uncommon
Onset of bleeding following trauma	delayed	immediate

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† Assistant Professor of Pathology, Department of Pathology, University of Kansas School of Medicine-Kansas City.

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TABLE II
SCREENING TESTS FOR HEMOSTATIC DISORDERS

<i>Vascular/Platelet disorders</i>	<i>Coagulation disorders</i>
Platelet count	Prothrombin time
Bleeding time (template)	Activated partial thromboplastin time
Blood film inspection	Thrombin time
Tourniquet test	Fibrinogen

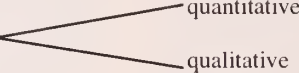
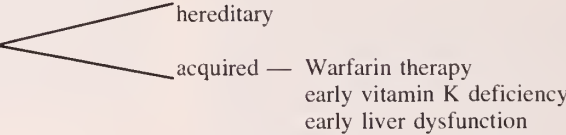
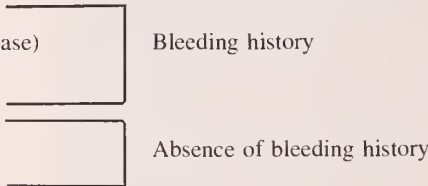
defects the platelet count and platelet morphology on peripheral blood smear are usually normal. Patients with a defect in platelet function may show similar results, in which case specific tests of platelet function — such as platelet aggregation studies — should be performed.

Spontaneous bleeding due to thrombocytopenia generally does not occur until the platelet count is below $40 \times 10^9/L$. At this level, the likelihood of bleeding is inversely proportional to the platelet

count, and at a count below $10 \times 10^9/L$, most patients will experience abnormal bleeding. Further investigations are necessary in the patient with a low platelet count, especially bone marrow examination. When bleeding occurs with a platelet count above $40 \times 10^9/L$, an abnormality of platelet function should be suspected. It is not unusual to have a defect of platelet function combined with thrombocytopenia, e.g. chronic idiopathic thrombocytopenic purpura.

Separate portions of the coagulation cascade are assessed by the major coagulation screening tests. The prothrombin time (PT) tests the extrinsic and common pathway, the activated partial thromboplastin time (APTT) tests the intrinsic and common pathway, and the thrombin time (TT) is a measure of the conversion of fibrinogen to fibrin. A common source of error is the contamination of blood samples by small amounts of heparin in indwelling catheters. It is important to exclude this as a possible cause for an abnormal test result before embarking on further investigations.

TABLE III
PT AND APTT AS SCREENING TESTS IN DISORDERS OF HEMOSTASIS

<i>PT</i>	<i>APTT</i>	<i>Possible Abnormality</i>
N	N	Platelet abnormality  Vascular abnormality XIII deficiency α -2-Antiplasmin deficiency
\uparrow	N	VII deficiency  acquired — Warfarin therapy early vitamin K deficiency early liver dysfunction
N	\uparrow	Hemophilia A Hemophilia B (Christmas disease) Von Willebrand's disease XI deficiency XII deficiency Deficiency of HMWK Deficiency of Prekallikrein Coagulation inhibitor Heparin 
\uparrow	\uparrow	X deficiency V deficiency Prothrombin deficiency Hypofibrinogenemia/dysfibrinogenemia Liver disease Vitamin K deficiency DIC Inhibitor (eg lupus anticoagulant) Warfarin therapy Heparin

If the PT and APTT are normal despite evidence of an abnormal bleeding tendency, and a platelet or vascular disorder have been excluded, the possibility of factor XIII deficiency should be entertained (*Table III*). Factor XIII deficiency can be specifically identified by demonstrating solubility of the plasma clot in 5 molar urea. Occasionally, patients with mild hemophilia A, B, or von Willebrand's disease have factor VIII or IX levels high enough to produce a normal APTT, but they may bleed excessively following surgery or trauma. Recently, patients have been described with a rare bleeding disorder due to hereditary alpha-2-antiplasmin deficiency, with subsequently increased fibrinolysis. Specific assays for alpha-2-antiplasmin levels are required for diagnosis, although shortening of the euglobulin lysis time may suggest this disorder.

Isolated prolongation of the PT with a normal APTT usually signifies deficiency of factor VII (*Table III*). Hereditary factor VII deficiency is relatively uncommon. Because of the short half-life of factor VII, early liver disease or vitamin K deficiency may initially manifest with prolongation of the PT.

A prolonged APTT with a normal PT indicates a defect of the intrinsic pathway. In the patient who has no abnormal bleeding history, deficiency of factor XII, prekallikrein (Fletcher factor), or high molecular weight kininogen (Fitzgerald factor) may be present, and a lupus anticoagulant should be considered. If a history of excessive bleeding is present, the possible causes include factor VIII deficiency (hemophilia or von Willebrand's disease), factor IX deficiency (Christmas disease), factor XI deficiency, coagulation inhibitors (other than the lupus anticoagulant), and heparin administration (*Table III*).

When both the PT and APTT are prolonged there are two major possibilities. Firstly, there may be a single defect of the common pathway such as a deficiency of factor X, factor V, prothrombin or

fibrinogen, or an abnormal fibrinogen molecule (dysfibrinogenemia). Secondly, and more commonly, multiple clotting factor defects may be present, such as may occur with liver disease, vitamin K deficiency, oral anticoagulants, disseminated intravascular coagulation (DIC), coagulation inhibitors or heparin administration (*Table III*). The screening tests are thus very useful for localizing defects to specific sites in the coagulation pathway, so that further assays for specific coagulation factors can be undertaken as required.

Coagulation Inhibitors

The most common coagulation inhibitors are factor VIII inhibitors, which occur most often in patients with hemophilia who have received multiple transfusions of factor VIII concentrates or other plasma products. Inhibitors directed against factor VIII or other sites in the coagulation pathway may occasionally be present in the postpartum period, during therapy with drugs such as penicillin or phenothiazines, in paraproteinemias, lymphoma, and collagen-vascular disorders such as rheumatoid arthritis and systemic lupus erythematosus. Idiopathic inhibitors occasionally occur in the older age group.

An inhibitor should be suspected whenever no obvious cause for a prolonged PT or APTT can be elicited on history or examination. The definitive diagnosis requires laboratory confirmation. A mixture is made of equal parts of patient's plasma and normal plasma, and the PT, or more usually the APTT, is measured immediately and repeated after a one hour incubation of the mixture at 37°C. When there is a clotting factor deficiency, mixing with normal plasma corrects the prolonged APTT, but in the presence of a coagulation inhibitor, the APTT remains prolonged (*Table IV*). Two types of inhibition may be seen. The immediate APTT measurement on the mixture may fail to correct to normal

TABLE IV
THE APTT IN THE DIAGNOSIS OF FACTOR DEFICIENCY OR COAGULATION INHIBITORS

	<i>Patient's plasma</i>	<i>Mixture of equal parts patient's + normal plasma</i>	
		<i>0 mins</i>	<i>60 mins</i>
Factor deficiency	↑	Correction	Correction
Inhibitor (immediate, eg heparin)	↑	↑	↑
Inhibitor (time-dependent eg anti-factor VIII)	↑	Correction (partial or complete)	↑

TABLE V
LABORATORY FEATURES OF DIC

Decreasing platelet count
Prolonged PT and APTT
Decreasing fibrinogen
Increased fibrin degradation products
Prolonged thrombin time
Positive protamine sulfate/ethanol gelation test for fibrin monomer
Decreased antithrombin III level
Decrease in factors V, VIII
Schistocytosis

(immediate inhibition), as seen, for example, with the lupus anticoagulant or heparin. Alternatively, the initial APTT corrects to normal, while the APTT is prolonged after one hour's incubation of the mixture (time dependent inhibition), which is typical of factor VIII inhibitors. Patients with a lupus anticoagulant typically do not have abnormal bleeding and in fact may present with a thrombotic tendency. This inhibitor is an *in vitro* phenomenon, directed against the coagulation complexes containing phospholipid.

Diagnosis of DIC

DIC should be suspected when bleeding from multiple sites occurs in the setting of one of the underlying disorders known to be associated with DIC. The diagnosis requires laboratory confirmation. Acute DIC is likely to manifest with the classical laboratory features (Table V), but these may not be present in subacute or chronic DIC.

An important pathophysiological mechanism in DIC is persistent or recurrent elaboration of excess amounts of thrombin within the circulation, associated with accelerated turnover of various coagulation factors as well as platelets. The levels of these factors depend on their relative rates of destruction and synthesis. The excess thrombin production results in increased conversion of fibrinogen to fibrin, and if this occurs at a faster rate than hepatic fibrinogen synthesis, the fibrinogen level will drop. Frequently, however, the liver produces increased amounts of fibrinogen in response to the underlying disease process, resulting in only a mild to moderate decrease in fibrinogen. Fibrinogen turnover studies, however, would be markedly abnormal. The action of circulating thrombin on fibrinogen results in the production of fibrin monomer, which tends to remain in a soluble form in the circulation in the presence of large fibrin degradation products. This

fibrin monomer can be detected by the protamine sulfate or ethanol gelation tests. A negative test, however, does not necessarily exclude DIC.

One of the earliest and most sensitive indicators of the presence of DIC is a drop in the platelet count; even a decrease within the normal range may be significant. Furthermore, platelet function may be impaired due to platelet activation and release reaction within the circulation, with consequent storage nucleotide depletion.

In most patients with DIC, the fibrinolytic system is secondarily activated, resulting in increased amounts of fibrin degradation products (FDP). When these products accumulate in excess, they further impair the hemostatic mechanism by inhibiting fibrin polymerization and the conversion of fibrinogen to fibrin, and inhibiting platelet function. Raised FDP levels are a relatively non-specific finding, however, since they may be increased in any situation where their clearance is decreased, such as renal failure and liver dysfunction, or where significant tissue breakdown has occurred, *e.g.* after surgery.

The PT and APTT may be prolonged in DIC due to hypofibrinogenemia, inhibition by FDP, or excess consumption of factors V, VIII, and prothrombin. The excess thrombin production results in consumption of antithrombin III, and its measurement provides a means of confirming the diagnosis and monitoring the course of DIC. The thrombin time may be prolonged due to a low fibrinogen level as well as the inhibitory effect of elevated FDPs on the conversion of fibrinogen to fibrin.

The formation of fibrin strands within the microvasculature may lead to a micro-angiopathic hemolytic state with schistocytosis noted on the peripheral blood smear. This is seen in less than 50 per cent of patients, however, and its presence is not necessarily correlated with the severity of the underlying DIC process.

Suggested Readings

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Editorial COMMENT

Physicians have had numerous successes to their credit in recent decades, but in one area — explaining and justifying their fees to the public — they have failed rather badly. Perhaps, considering the accessory events and the medical temperament generally, it couldn't have been otherwise but the efforts have been, at best, ineffectual.

To a large degree, the successes themselves have been more than a casual factor in the matter. The accomplishments mean that more people are receiving more medical care than at any time in history and with an overall degree of success never before known. But the public has never understood the full content of the medical fee. It is titillated by the dramatic front-page stories of transplants and test tubes and momentarily impressed by the reports of great cost which, of course, have no reality since it is not involved directly. But the more routine fees covering some immediate service (and which, although it doesn't see the connection, support a system that makes such successes possible), it does see — and doesn't like.

Complaints about fees are not new — every age has recorded its annoyance at physicians' charges. The current situation reflects a deeper change in the public-profession relationship stemming from both intrinsic medical changes and the socio-economic climate. Physicians, under the weight of tradition, have been accustomed to making diagnoses, prescribing therapies, and controlling management with full authority. Patients have (usually) complied and recovered with sufficient frequency to indicate that the system was valid. And the physician determined the charge — ranging from nothing to a more than ample sum depending upon individual factors but still, in the final determination, the physician's estimate of its worth. Custom and an unspoken competition did the rest.

The physician's attitude was born of this certainty of professional assessment plus a sense of integrity and ethical purpose which simply ignored external influences, both of which are undergoing a process of profound readjustment or even discard. Moreov-

er, there was no easy translation of the medical service into material market-place terms (which is really what is going on now). The various pressures for change were at first not so much resisted as ignored — the service was too important to think it could be seriously assaulted by such intrusions. Warnings abounded and at first they were only an irritation, but as medical positions were increasingly eroded and financial battles were lost, the profession's developing efforts to regain a semblance of control have come across to the public as declarations of self-interest and to the membership as reactive rather than innovative.

The current crop of efforts applies less to physician remuneration directly than to other areas of medical service, but the direct control of physicians' charges is presaged. One of the prime social efforts has been to get physicians' fees publicized. It might seem a bold, even — at this point — heretical suggestion but perhaps the profession should go one step farther and present patients with a very detailed break-down of the substance of their charges related to costs. At least, it might bring into focus those chronic over-chargers who contribute to the profession's bad financial name.

So the profession hasn't appreciated what the public wanted and the public hasn't understood the entirety of what is involved in medical fees. Medical merchandising (we might as well call it that) has not been pitched properly to a public accustomed to a different form of commercial contact. Medicine's approach really is changing, however. Several "emergency centers" have appeared in the Golden City. The morning paper carried an advertisement for one that gives the name of "the winner of our drawing for one free year of medical service for her family." In the interests of cost containment, we presume the expenses incurred in providing such care will be transferred to the advertising (read public relations) budget in the manner of the costs of promoting Popsy-Whamos Cereal or Gloppy Burgers. Now *that* the public can understand. — D.E.G.

**New study reveals
no interaction between**



Ativan® (lorazepam) and Darvon® (propoxyphene HCl) ©

In a study evaluating the influence of propoxyphene coadministration on the pharmacokinetics of the oxidatively metabolized benzodiazepines Xanax® (alprazolam) © and Valium® (diazepam) ©, and a benzodiazepine metabolized by conjugation, Ativan® (lorazepam), the following results were reported:

with Xanax, propoxyphene caused a large and highly significant prolongation of half-life and impairment of total metabolic clearance.¹

in the case of Valium, propoxyphene produced a small but not statistically significant impairment of clearance.¹

propoxyphene had no apparent effect on the distribution, half-life or clearance of Ativan.¹

In this randomized crossover study, eight healthy male and female volunteers received single oral doses of alprazolam (1 mg), six received single IV doses of diazepam (10 mg), and five received single IV doses of lorazepam (2 mg), once in a drug-free control state and again during coadministration of pro-

poxyphene (65 mg q6h). Consistent with previous findings, this study evidences that Ativan does not interact with drugs that undergo oxidative metabolism.²⁻⁵ In contrast to most other benzodiazepines, Ativan does not compete for the cytochrome P-450 enzyme system.

The clinical implications of the pharmacokinetic interaction, or non-interaction, of propoxyphene with benzodiazepines are not established by this study. Even without a pharmacokinetic interaction, propoxyphene and benzodiazepines share central depressant properties and therefore should be coadministered with suitable caution. A concurrent pharmacokinetic interaction indicates a need for even further caution. Coadministration of propoxyphene and alprazolam, for example, would produce not only the expected pharmacodynamic interaction, but also whatever additional central depressant effect would be produced by the elevated steady-state plasma concentrations of alprazolam due to its impaired clearance.

Caution should also be observed when propoxyphene is prescribed for patients who use alcohol to excess.

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for (lorazepam) ©
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Brief Summary of Prescribing Information.

Indications and Usage: Management of anxiety disorders or short-term relief of symptoms of anxiety or anxiety associated with depressive symptoms. Anxiety or tension associated with stress of everyday life usually does not require treatment with an anxiolytic. Effectiveness in long-term use, i.e., more than 4 months, has not been assessed by systematic clinical studies. Reassess periodically usefulness of the drug for the individual patient.

Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma.

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any antianxiety agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown, but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper GI disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia, some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chloridiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Transient amnesia or memory impairment has been reported in association with the use of benzodiazepines.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levaterenol Bitartrate Injection USP. Usefulness of dialysis has not been determined.

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DOSAGE: Individualize for maximum beneficial effects. Increase dose gradually when needed, giving higher evening dose before increasing daytime doses. Anxiety, usually 2-3mg/day given b.i.d. or t.i.d.; dosage may vary from 1 to 10mg/day in divided doses. For elderly or debilitated, initially 1-2mg/day; insomnia due to anxiety or transient situational stress, 2-4mg h.s.

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Warnings: Peptic ulceration and GI bleeding, sometimes severe, have been reported. Ulceration, perforation and bleeding may end fatally. An association has not been established. Use *Motrin* Tablets under close supervision in patients with a history of upper gastrointestinal tract disease, after consulting ADVERSE REACTIONS. In patients with active peptic ulcer and active rheumatoid arthritis, try nonulcerogenic drugs, such as gold. If *Motrin* Tablets are used, observe the patient closely for signs of ulcer perforation or GI bleeding.

Chronic studies in rats and monkeys have shown mild renal toxicity with papillary edema and necrosis. Renal papillary necrosis has rarely been shown in humans treated with *Motrin* Tablets.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue *Motrin* Tablets and the patient should have an ophthalmologic examination, including central visual fields and color vision testing.

Fluid retention and edema have been associated with *Motrin* Tablets; use with caution in patients with a history of cardiac decompensation or hypertension. In patients with renal impairment, reduced dosage may be necessary. Prospective studies of *Motrin* Tablets safety in patients with chronic renal failure have not been done.

Motrin Tablets can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, skin rash, weight gain, or edema.

Patients on prolonged corticosteroid therapy should have therapy tapered slowly when *Motrin* Tablets are added.

The antipyretic, anti-inflammatory activity of *Motrin* Tablets may mask inflammation and fever.

As with other nonsteroidal anti-inflammatory drugs, borderline elevations of liver tests may occur in up to 15% of patients. These abnormalities may progress, may remain essentially unchanged, or may be transient with continued therapy. Meaningful elevations of SGPT or SGOT (AST) occurred in controlled clinical trials in less than 1% of patients. Severe hepatic reactions, including jaundice and cases of fatal hepatitis, have been reported with ibuprofen as with other nonsteroidal anti-inflammatory drugs. If liver disease develops or if systemic manifestations occur (e.g. eosinophilia, rash, etc.), *Motrin* should be discontinued.

Drug interactions. Aspirin: used concomitantly may decrease *Motrin* blood levels.

Coumarin: bleeding has been reported in patients taking *Motrin* and coumarin.

Pregnancy and nursing mothers: *Motrin* should not be taken during pregnancy or by nursing mothers.

Adverse Reactions: The most frequent type of adverse reaction occurring with *Motrin* is gastrointestinal of which one or more occurred in 4% to 16% of the patients.

Incidence Greater than 1% (but less than 3%)—Probable Causal Relationship

Gastrointestinal: Nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence). **Central Nervous System:** Dizziness,* headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic/Endocrine:** Decreased appetite. **Cardiovascular:** Edema, fluid retention (generally responds promptly to drug discontinuation; see PRECAUTIONS).

Incidence less than 1%—Probable Causal Relationship**

Gastrointestinal: Gastric or duodenal ulcer with bleeding and/or perforation, gastrointestinal hemorrhage, melena, gastritis, hepatitis, jaundice, abnormal liver function tests. **Central Nervous System:** Depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome, alopecia. **Special Senses:** Hearing loss, amblyopia (blurred and/or diminished vision, scotomata, and/or changes in color vision) (see PRECAUTIONS). **Hematologic:** Neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs positive), thrombocytopenia with or without purpura, eosinophilia, decreases in hemoglobin and hematocrit. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure, palpitations. **Allergic:** Syndrome of abdominal pain, fever, chills, nausea and vomiting, anaphylaxis, bronchospasm (see CONTRAINDICATIONS). **Renal:** Acute renal failure in patients with pre-existing significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria. **Miscellaneous:** Dry eyes and mouth, gingival ulcer, rhinitis.

Incidence less than 1%—Causal Relationship Unknown**

Gastrointestinal: Pancreatitis. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri. **Dermatologic:** Toxic epidermal necrolysis, photoallergic skin reactions. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Bleeding episodes (e.g., epistaxis, menorrhagia). **Metabolic/Endocrine:** Gynecomastia, hypoglycemic reaction. **Cardiovascular:** Arrhythmias (sinus tachycardia, sinus bradycardia). **Allergic:** Serum sickness, lupus erythematosus syndrome, Henoch-Schönlein vasculitis. **Renal:** Renal papillary necrosis.

*Reactions occurring in 3% to 9% of patients treated with *Motrin*. (Those reactions occurring in less than 3% of the patients are unmarked.)

**Reactions are classified under "Probable Causal Relationship (PCR)" if there has been one positive rechallenge or if three or more cases occur which might be causally related. Reactions are classified under "Causal Relationship Unknown" if seven or more events have been reported but the criteria for PCR have not been met.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine so alkaline diuresis may be beneficial.

Dosage and Administration: Rheumatoid arthritis and osteoarthritis. Suggested dosage is 300, 400, or 600 mg t.i.d. or q.i.d. Do not exceed 2400 mg per day. Mild to moderate pain: 400 mg every 4 to 6 hours as necessary.

Caution: Federal law prohibits dispensing without prescription.

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Endometriosis

(Continued from page 268)

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Chronic Pain

(Continued from page 266)

thus are accessible to treatment by needle, pressure, or electrical stimulation.

Within the past ten years transcutaneous electrical nerve stimulation (TENS) has had a wide trial and considerable acceptance as a procedure to ameliorate pain. An ingeniously compact, portable, battery-powered apparatus delivers a modulated electrical current by means of electrodes applied to the body. Electrode placement is largely a matter of trial and error. The stimulus is more effective when the frequency, intensity, and wave form is individualized in each case.

In the early 1970s surgeons were implanting these electrodes in the spinal cord in the belief that this would provide greater relief. The procedure proved to be no more effective than the simpler transcutaneous application and has been abandoned by most practitioners. Again, claims for pain relief vary from no effect to enthusiastic testimonials for a high incidence of success.

Psychologists and psychiatrists have yet another approach to pain control. Both professionals use basic behavior modification techniques varying in depth and complexity. Patients who cannot be taught to ignore the pain are guided into accepting and adapting to it.

There are countless other ways to attack the problem of pain control, and without doubt many more will be developed. It is obvious that no one approach is clearly more effective than another. Every procedure works on some people in some cases. Every procedure also fails on a number of people.

To handle pain with any hope of success, two important facts must be considered. First, the perception and appreciation of pain is ultimately an emotional reaction. As such, the feeling is completely vulnerable to psychological influences. Cultural background, suggestion, attention, current environmental conditions, motivation, all play a role in shaping thoughts and emotions including the complaint of "pain."

The other consideration concerns a truism by Hughlings Jackson in the mid 1800s. Simply stated, the central nervous system cannot consciously

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appreciate two simultaneous stimuli. The stronger will dampen or abolish the weaker. This could very well explain why acupuncture, transcutaneous stimulation, applications of cold or heat, massage, and countless other procedures can all be effective in some instances. When a stimulus — whatever it may be — is more intense than the feeling of persistent pain, the higher nerve centers become occupied with this more acceptable emotion and the patient feels relieved of his pain. The answer to successful management of pain may not be found in newer, more complicated gadgetry, pseudo-scientific approaches, and complex hypotheses; we may simply need more logical and appropriate applications of the proven agents already available.

References are available from Dr. Novak, Colmery-O'Neil Veterans Administration Medical Center, 2200 Gage Blvd., Topeka KS 66622.

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Precautions: In elderly and debilitated patients, it is recommended that the dosage be limited to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in severely depressed patients, or in those with latent depression or suicidal tendencies, or in those with impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, light-headedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins, and alkaline phosphatase; and paradoxical reactions, e.g., excitement, stimulation and hyperactivity.

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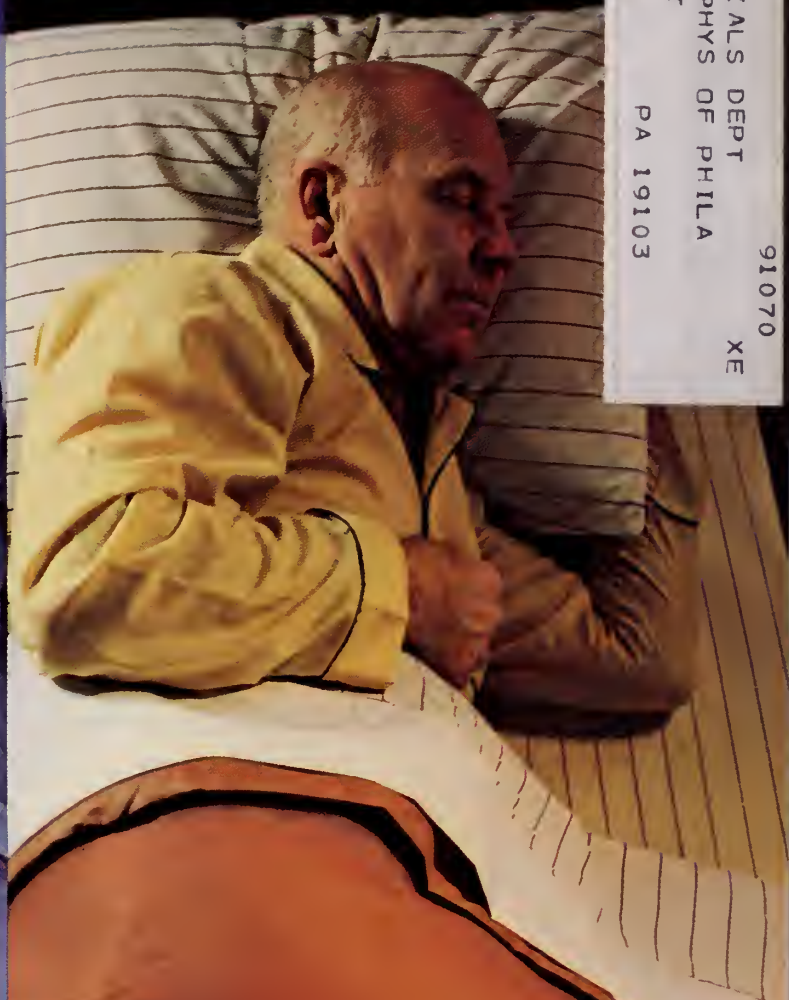
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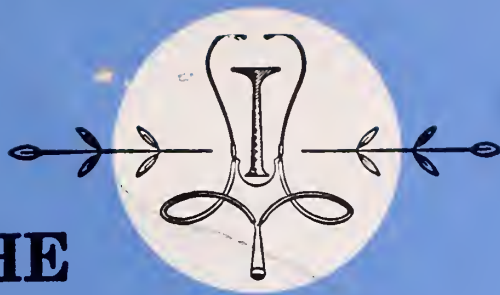
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Kansas Medicine

(The Journal of the Kansas Medical Society)

With the January 1985 issue, KANSAS MEDICINE (*The Journal of the Kansas Medical Society*) will make available a Physicians' Directory section to provide members of the Kansas Medical Society with a means of identifying themselves and their services to their colleagues.

The following information sets forth the conditions for inclusion of material in the directory both as to content and charges. If there are particular questions beyond the information herein, please contact the Managing Editor at the JOURNAL office, 1300 Topeka Avenue, Topeka KS 66612; or call 913-235-2383.

Content

1. Directory listings will be sold on the basis of page-inch (or multiples). This means that the format will extend across the page and one inch (or more) in depth.
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3. Listings by name will be limited to members of the Kansas Medical Society.
4. Groups bearing association or corporate names must include in the text the names of those individuals who are members of the Kansas Medical Society.
5. Individuals in a group who are not members will not be listed individually, but if their presence in the group accounts for the availability of a special service by the group, this service may be noted elsewhere in the notice.
6. Information considered suitable for these notices may include type of practice. If a specialty is involved, certification may be duly included; if an individual is not certified in a given specialty, adequate justification for inclusion of such a designation must be available. Directory listings may include information regarding conditions of professional practice such as Blue Shield participation and acceptance of Medicare assignment.
7. Identification of specialty or practice limitations will generally be related to those categories utilized in the annual Membership Directory of the Kansas Medical Society, although multiple classifications (not available in the annual Membership Directory) may be included.
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In a study evaluating the influence of propoxyphene coadministration on the pharmacokinetics of the oxidatively metabolized benzodiazepines Xanax[®] (alprazolam) [Ⓢ] and Valium[®] (diazepam) [Ⓢ], and a benzodiazepine metabolized by conjugation, Ativan[®] (lorazepam), the following results were reported:

with Xanax, propoxyphene caused a large and highly significant prolongation of half-life and impairment of total metabolic clearance.¹

in the case of Valium, propoxyphene produced a small but not statistically significant impairment of clearance.¹

propoxyphene had no apparent effect on the distribution, half-life or clearance of Ativan.¹

In this randomized crossover study, eight healthy male and female volunteers received single oral doses of alprazolam (1 mg), six received single IV doses of diazepam (10 mg), and five received single IV doses of lorazepam (2 mg), once in a drug-free control state and again during coadministration of pro-

poxyphene (65 mg q6h). Consistent with previous findings, this study evidences that Ativan does not interact with drugs that undergo oxidative metabolism.²⁻⁵ In contrast to most other benzodiazepines, Ativan does not compete for the cytochrome P-450 enzyme system.

The clinical implications of the pharmacokinetic interaction, or non-interaction, of propoxyphene with benzodiazepines are not established by this study. Even without a pharmacokinetic interaction, propoxyphene and benzodiazepines share central depressant properties and therefore should be coadministered with suitable caution. A concurrent pharmacokinetic interaction indicates a need for even further caution. Coadministration of propoxyphene and alprazolam, for example, would produce not only the expected pharmacodynamic interaction, but also whatever additional central depressant effect would be produced by the elevated steady-state plasma concentrations of alprazolam due to its impaired clearance.

Caution should also be observed when propoxyphene is prescribed for patients who use alcohol to excess.

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Contraindications: Known sensitivity to benzodiazepines or acute narrow-angle glaucoma

Warnings: Not recommended in primary depressive disorders or psychoses. As with all CNS-acting drugs, warn patients not to operate machinery or motor vehicles, and of diminished tolerance for alcohol and other CNS depressants.

Physical and Psychological Dependence: Withdrawal symptoms like those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepines (including convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Addiction-prone individuals, e.g. drug addicts and alcoholics, should be under careful surveillance when on benzodiazepines because of their predisposition to habituation and dependence. Withdrawal symptoms have also been reported following abrupt discontinuance of benzodiazepines taken continuously at therapeutic levels for several months.

Precautions: In depression accompanying anxiety, consider possibility for suicide.

For elderly or debilitated patients, initial daily dosage should not exceed 2mg to avoid oversedation. Terminate dosage gradually since abrupt withdrawal of any anxiolytic agent may result in symptoms like those being treated: anxiety, agitation, irritability, tension, insomnia and occasional convulsions. Observe usual precautions with impaired renal or hepatic function. Where gastrointestinal or cardiovascular disorders coexist with anxiety, note that lorazepam has not been shown of significant benefit in treating gastrointestinal or cardiovascular component. Esophageal dilation occurred in rats treated with lorazepam for more than 1 year at 6mg/kg/day. No effect dose was 1.25mg/kg/day (about 6 times maximum human therapeutic dose of 10mg/day). Effect was reversible only when treatment was withdrawn within 2 months of first observation. Clinical significance is unknown; but use of lorazepam for prolonged periods and in geriatrics requires caution and frequent monitoring for symptoms of upper GI disease. Safety and effectiveness in children under 12 years have not been established.

ESSENTIAL LABORATORY TESTS: Some patients have developed leukopenia; some have had elevations of LDH. As with other benzodiazepines, periodic blood counts and liver function tests are recommended during long-term therapy.

CLINICALLY SIGNIFICANT DRUG INTERACTIONS: Benzodiazepines produce CNS depressant effects when administered with such medications as barbiturates or alcohol.

CARCINOGENESIS AND MUTAGENESIS: No evidence of carcinogenic potential emerged in rats during an 18-month study. No studies regarding mutagenesis have been performed.

PREGNANCY: Reproductive studies were performed in mice, rats, and 2 strains of rabbits. Occasional anomalies (reduction of tarsals, tibia, metatarsals, malrotated limbs, gastroschisis, malformed skull and microphthalmia) were seen in drug-treated rabbits without relationship to dosage. Although all these anomalies were not present in the concurrent control group, they have been reported to occur randomly in historical controls. At 40mg/kg and higher, there was evidence of fetal resorption and increased fetal loss in rabbits which was not seen at lower doses. Clinical significance of these findings is not known. However, increased risk of congenital malformations associated with use of minor tranquilizers (chloridiazepoxide, diazepam and meprobamate) during first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, use of lorazepam during this period should almost always be avoided. Possibility that a woman of child-bearing potential may be pregnant at institution of therapy should be considered. Advise patients if they become pregnant to communicate with their physician about desirability of discontinuing the drug. In humans, blood levels from umbilical cord blood indicate placental transfer of lorazepam and its glucuronide.

NURSING MOTHERS: It is not known if oral lorazepam is excreted in human milk like other benzodiazepines. As a general rule, nursing should not be undertaken while on a drug since many drugs are excreted in milk.

Adverse Reactions, if they occur, are usually observed at beginning of therapy and generally disappear on continued medication or on decreasing dose. In a sample of about 3,500 anxious patients, most frequent adverse reaction is sedation (15.9%), followed by dizziness (6.9%), weakness (4.2%) and unsteadiness (3.4%). Less frequent are disorientation, depression, nausea, change in appetite, headache, sleep disturbance, agitation, dermatological symptoms, eye function disturbance, various gastrointestinal symptoms and autonomic manifestations. Incidence of sedation and unsteadiness increased with age. Small decreases in blood pressure have been noted but are not clinically significant, probably being related to relief of anxiety.

Transient amnesia or memory impairment has been reported in association with the use of benzodiazepines.

Overdosage: In management of overdosage with any drug, bear in mind multiple agents may have been taken. Manifestations of overdosage include somnolence, confusion and coma. Induce vomiting and/or undertake gastric lavage followed by general supportive care, monitoring vital signs and close observation. Hypotension, though unlikely, usually may be controlled with Levartenerol Bitartrate Injection U.S.P. Usefulness of dialysis has not been determined.

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Contraindications: Anaphylactoid reactions have occurred in individuals hypersensitive to Motrin Tablets or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin, iodides, or other nonsteroidal anti-inflammatory agents

Warnings: Peptic ulceration and GI bleeding, sometimes severe, have been reported. Ulceration, perforation and bleeding may end fatally. An association has not been established. Use Motrin Tablets under close supervision in patients with a history of upper gastrointestinal tract disease, after consulting ADVERSE REACTIONS. In patients with active peptic ulcer and active rheumatoid arthritis, try nonulcerogenic drugs, such as gold. If Motrin Tablets are used, observe the patient closely for signs of ulcer perforation or GI bleeding.

Chronic studies in rats and monkeys have shown mild renal toxicity with papillary edema and necrosis. Renal papillary necrosis has rarely been shown in humans treated with Motrin Tablets.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin Tablets and the patient should have an ophthalmologic examination, including central visual fields and color vision testing.

Fluid retention and edema have been associated with Motrin Tablets, use with caution in patients with a history of cardiac decompensation or hypertension. In patients with renal impairment, reduced dosage may be necessary. Prospective studies of Motrin Tablets safety in patients with chronic renal failure have not been done.

Motrin Tablets can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, skin rash, weight gain, or edema.

Patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin Tablets are added.

The antipyretic, anti-inflammatory activity of Motrin Tablets may mask inflammation and fever.

As with other nonsteroidal anti-inflammatory drugs, borderline elevations of liver tests may occur in up to 15% of patients. These abnormalities may progress, may remain essentially unchanged, or may be transient with continued therapy. Meaningful elevations of SGPT or SGOT (AST) occurred in controlled clinical trials in less than 1% of patients. Severe hepatic reactions, including jaundice and cases of fatal hepatitis, have been reported with ibuprofen as with other nonsteroidal anti-inflammatory drugs. If liver disease develops or if systemic manifestations occur (e.g. eosinophilia, rash, etc.), Motrin should be discontinued.

Drug interactions. Aspirin: used concomitantly may decrease Motrin blood levels.

Coumarin: bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy or by nursing mothers.

Adverse Reactions: The most frequent type of adverse reaction occurring with Motrin is gastrointestinal of which one or more occurred in 4% to 16% of the patients.

Incidence Greater than 1% (but less than 3%)—Probable Causal Relationship

Gastrointestinal: Nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence). **Central Nervous System:** Dizziness,* headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic/Endocrine:** Decreased appetite. **Cardiovascular:** Edema, fluid retention (generally responds promptly to drug discontinuation; see PRECAUTIONS).

Incidence less than 1%—Probable Causal Relationship**

Gastrointestinal: Gastric or duodenal ulcer with bleeding and/or perforation, gastrointestinal hemorrhage, melena, gastritis, hepatitis, jaundice, abnormal liver function tests. **Central Nervous System:** Depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome, alopecia. **Special Senses:** Hearing loss, amblyopia (blurred and/or diminished vision, scotomata, and/or changes in color vision) (see PRECAUTIONS). **Hematologic:** Neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs positive), thrombocytopenia with or without purpura, eosinophilia, decreases in hemoglobin and hematocrit. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure, palpitations. **Allergic:** Syndrome of abdominal pain, fever, chills, nausea and vomiting; anaphylaxis, bronchospasm (see CONTRAINDICATIONS). **Renal:** Acute renal failure in patients with pre-existing significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria. **Miscellaneous:** Dry eyes and mouth, gingival ulcer, rhinitis.

Incidence less than 1%—Causal Relationship Unknown**

Gastrointestinal: Pancreatitis. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri. **Dermatologic:** Toxic epidermal necrolysis, photoallergic skin reactions. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Bleeding episodes (e.g., epistaxis, menorrhagia). **Metabolic/Endocrine:** Gynecomastia, hypoglycemic reaction. **Cardiovascular:** Arrhythmias (sinus tachycardia, sinus bradycardia). **Allergic:** Serum sickness, lupus erythematosus syndrome, Henoch-Schonlein vasculitis. **Renal:** Renal papillary necrosis.

*Reactions occurring in 3% to 9% of patients treated with Motrin. (Those reactions occurring in less than 3% of the patients are unmarked.)

**Reactions are classified under "Probable Causal Relationship (PCR)" if there has been one positive rechallenge or if three or more cases occur which might be causally related. Reactions are classified under "Causal Relationship Unknown" if seven or more events have been reported but the criteria for PCR have not been met.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine so alkaline diuresis may be beneficial.

Dosage and Administration: Rheumatoid arthritis and osteoarthritis. Suggested dosage is 300, 400, or 600 mg t.i.d. or q.i.d. Do not exceed 2400 mg per day. Mild to moderate pain: 400 mg every 4 to 6 hours as necessary.

Caution: Federal law prohibits dispensing without prescription.

MED B 7:5

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- ☐ 2 Do you need *immediate* tax write-offs through proper tax planning?
- ☐ 3 Would you consider changing some business procedures to cut *future* taxes?
- ☐ 4 Are you setting up or changing estate plans for yourself or someone else?
- ☐ 5 Is your business required to file financial reports — anything from a statement of earnings to full-scale audited financial statements?
- ☐ 6 Do you feel it might be time to incorporate your business?
- ☐ 7 Are you preparing a business loan application — and need supporting documentation?
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- ☐ 9 Are you thinking of starting a pension or profit-sharing plan?
- ☐ 10 Do you need an *independent* source of professional management advice on how to use your resources more efficiently or how to make financial projections?

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Kansas Society of
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Council Meeting

Report of Meeting Held September 8, 1984

The Council convened September 8, 1984, at the Holiday Inn Holidome, Lawrence, beginning at 9:30 A.M. Present were: F. Calvin Bigler, M.D., President; Carl Ambler, M.D.; Stuart Averill, M.D.; James Bridgens, M.D.; David Clark, M.D.; Rex R. Fischer, M.D.; Herbert Fransen, M.D.; Jim Gleason, M.D.; Robert Haskins, M.D.; D. W. Hatton, M.D.; Martha Hunt; David A Leitch, M.D.; James A. Loeffler, M.D.; J. E. Lungstrum, M.D.; Wendale E. McAllaster, M.D.; Warren E. Meyer, M.D.; Steven Myrick, M.D.; John R. Neuen-schwander, M.D.; Forrest Pommerenke, M.D.; Lew W. Purinton, M.D.; E. D. Rathbun, M.D.; Ralph Reed, M.D.; Ann Rempel; Clifton Schopf, M.D.; Alex Scott, M.D.; Joan Sehdev, M.D.; R. A. Siemens, M.D.; Phillip Sisk, M.D.; R. M. Skibba, M.D.; Thomas F. Taylor, M.D.; Marijo Teare, Max Teare, M.D.; Jack Walker, M.D.; Linda Warren, M.D.; Roger Warren, M.D.; Wallace N. Weber, M.D.; Kermit G. Wedel, M.D.; Emerson D. Yoder, M.D.

Staff present: Jerry Slaughter, Val Braun and Gary Caruthers.

Dr. Bigler convened the meeting. He welcomed the members and invited Dr. Warren Meyer to present a thought for the day.

Budget

The 1985 dues, as previously approved, will be \$220. The Council also approved a two-year capital improvement plan for the Executive Office Building; an increase in certain staff salaries; an increase in the *Journal* printing cost allocation; and a reduction in the overall travel expenses.

Professional Liability

Approved that every dues paying member be billed a minimum of \$100 for the PL program and that voluntary contributions of greater amounts be recognized in a special manner. The funds will be utilized in the two-year KMS Action Plan, a legislative effort to improve the professional liability situation in Kansas. The envisioned legislation will be aimed at limiting attorneys' fees; limiting awards; removing the collateral source rule; and perhaps providing an innovative and quick way to resolve claims. The plan also includes an informational program designed to make the public and the legislature aware of the unacceptable professional liability

climate and its effects on health costs, patient care, and access to a comprehensive range of physician services.

Medical Records

The importance of reviewing the records prior to transfer of information was stressed. The Council then adopted the following as a guideline for physicians when filling requests by non-physicians for medical records:

Member physicians are frequently requested to supply copies of charts, medical records and other information to insurance companies, attorneys and other non-physicians. The provision of such material is a service being provided by the physician for which s/he should be compensated by the party requesting the records.

The charge imposed by the physician should properly include several components. Among these are:

- expenses involved in copying the records;
- reimbursement for the physician's time in reviewing the request and identifying the records;
- clerical time in reproducing the records.

It is the opinion of Legal Counsel that such costs can be billed either as a flat fee plus a charge per page for copying, or the copying charge may be established at a level sufficient to include the above items.

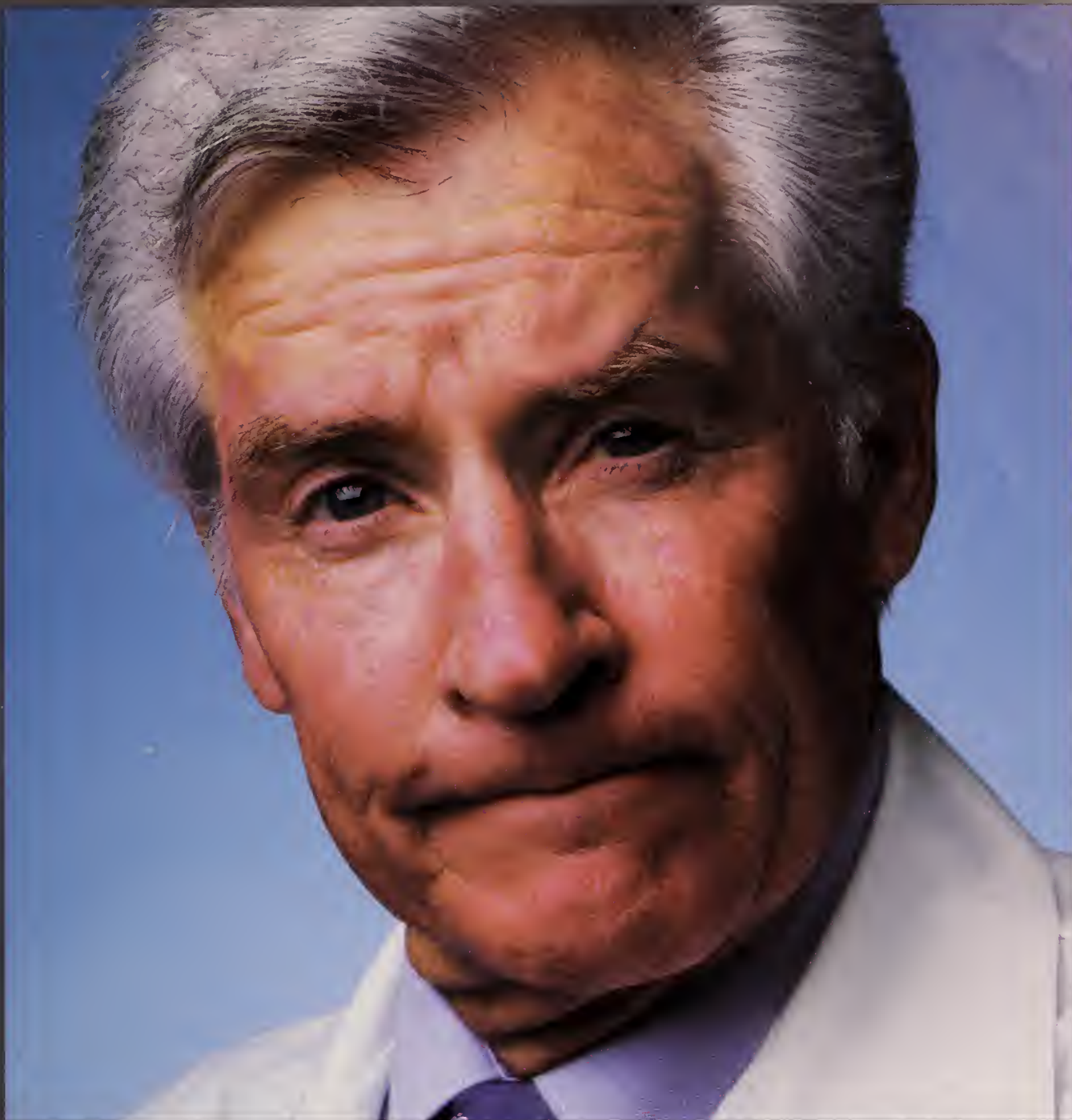
If a report, summary, or other additional document must be completed by the physician, then a fee representing a professional charge for this service may properly be requested.

KFMC

In response to Resolutions 84-21 and 84-31, the Council authorized establishing a better staff liaison between the Kansas Medical Society and the Kansas Foundation for Medical Care. A special ad hoc committee was charged with the responsibility of studying the issue and presenting a report at the January 19, 1985 meeting of this Council. The committee — chaired by Thomas Taylor, M.D., Salina — will consist of Drs. David Leitch, Garnett; Lew W. Purinton, M.D., Wichita; Edwin D. Rathbun, M.D., Liberal; Ralph Reed, M.D., Lawrence; and Alex Scott, M.D., Junction City.

KMS Journal

The Council heard a number of complaints from member physicians directed at the results of the
(Continued on page 289)



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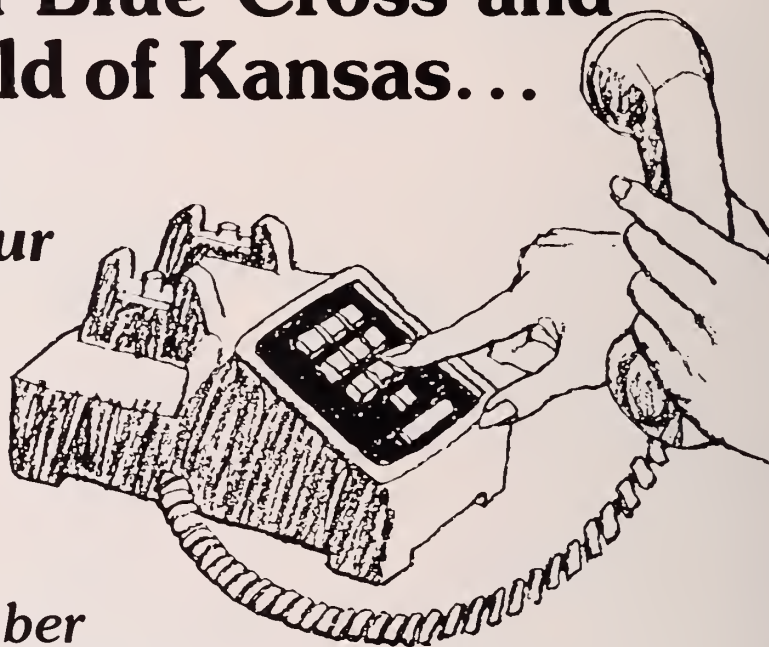
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Council Meeting

(Continued from page 286)

austerity program which reduced the *Journal's* format considerably. The Council accepted the editorial board report and authorized an increase in the size of the *Journal*; a change in cover and interior format; the change in the name to *KANSAS MEDICINE*; initiation of professional directory ads to be confined within the scope of professional ethics; discontinuance of special UKSM issues in favor of ongoing monthly series of featured papers, and incorporation of the *Newsletter* into the *Journal*.

Medicare Changes

October 1, 1984 is the deadline for physicians to determine their participation in the Medicare program. The AMA's lawsuit seeking a ruling on the constitutionality of the Medicare law changes does not relieve the physicians of the obligation of meeting that deadline. Participation in the Medicare program will not violate the "most favored nation" clause of the Blue Shield participating contract. Statistically, some 50 per cent of all claims and 80 per cent of all dollar amounts paid are assigned. At least 18 per cent of physicians always take assignment, while 30 per cent never do, with the remainder accepting assignment on a selected basis.

KaMPAC

The Council agreed to determine on an individual basis whether a financial contribution by KaMPAC to a political candidate carries with it the endorsement by the Kansas Medical Society. The Council briefly heard from the following guests: Jack Walker, M.D., candidate for State Senate; Forrest Pommerehke, M.D., member of Kansas State Board of Healing Arts; Ann Rempel (John), KMS Auxiliary President; Marijo Teare, Medical Student Section Representative.

KMS Insurance Committee was authorized to review the final insurance proposal which, if approved, will be an alternative to the existing Blue Cross/Blue Shield plan.

By-Laws Changes will be considered at the January Council meeting. Each Councilor will be mailed a copy of the current By-Laws, along with proposed changes for review prior to the meeting.

KMS-KU Liaison Committee has considered the following issues: competition for patients at primary care level; need for adequate number of patients to properly train students; quality of student education, particularly at the basic science level.

Smoking Prohibition

Resolution 84-10 called for prohibition of smoking at all formal KMS meetings. The following definition of "formal meeting" was adopted by the Council:

"Any meeting convened to conduct business of the Kansas Medical Society at which an agenda is used and minutes are kept."

Chiropractic

The Council agreed to postpone the study of Resolution 84-22, calling for a legislative study to define the scope of chiropractic practice.

Litigation Update

KMS has filed an *amicus curiae* brief on the issue

of collateral source benefit in a Wichita suit, hoping that the lower court opinion on the admissibility of such benefits will be upheld. KMS is monitoring the case involving several Emporia chiropractors who have sued the local school board for refusal to allow them to perform school physicals and use the term "physician."

AMA Nominations

The following candidacies of KMS members to AMA offices were announced: Roger Warren, M.D., AMPAC Board of Directors; William J. Reals, M.D., Council on Medical Education; Linda Warren, M.D., Ad Hoc Committee on Young Physicians; Alex Scott, M.D., Council on Long Range Planning.

The meeting adjourned at 11:30 A.M.

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Course Description: Paul Wehrle, M.D., President of the American Academy of Pediatrics, will be the keynote speaker. Outstanding faculty from three medical schools will join Dr. Wehrle in presenting topics including Infectious Diseases, Pediatric Orthopedics, Dermatology, Endocrinology and Pediatric and Adult Cardiology.

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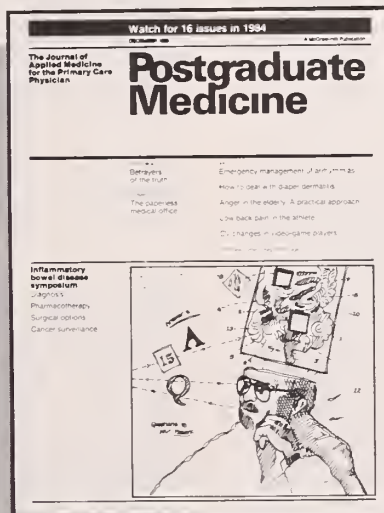


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GI Bleeding Caused by Amyloidosis

W. G. CAUBLE, M.D. *Wichita*

GASTROINTESTINAL BLEEDING can be caused by many pathological conditions — some may be quite rare and unusual and some may be difficult to diagnose. Amyloidosis may be a rare cause of this type of bleeding, and reported here is a case that was diagnosed as amyloidosis of the right colon associated with amyloidosis of the kidneys and prostate.

Case Report

A 69-year-old white male had experienced several bouts of intestinal bleeding. He was quite concerned and wished to avoid repeated hospitalization for bleeding. In October 1982, selective superior mesenteric angiogram had yielded negative results. An isotope scan for gastrointestinal bleeding was also negative. He had been hospitalized four times during 1983 with bleeding episodes.

The diagnosis of amyloidosis was first made in July 1974 when a biopsy of the left kidney was performed in California. A diagnosis of duodenal ulcer was made by endoscopy in February 1982. The following May, he developed kidney failure, and a study of tissue removed during transurethral resection revealed sclerosis with positive amyloid tissue. He was placed on home dialysis in June 1981. When first seen by the author, he had a peritoneal dialysis tube in place. He had been colonoscoped by another physician who was of the opinion that the bleeding was coming from hemorrhagic areas in the right colon. Two biopsies revealed polypoid leiomyoma and polypoid adenoma.

In October 1983, the patient's peritoneal dialysis tube was removed and a right colon resection was performed. A catheter was placed in the bladder, but he passed no urine during the surgery. During his convalescence, he progressed satisfactorily, and was on hemodialysis. He has since been hospitalized twice — in December 1983 for aortic stenosis and in March 1984 for congestive heart failure. He experienced no more bouts of gastrointestinal bleeding, but died May 6, 1984, after requesting that dialysis be discontinued approximately three weeks earlier.

The gross findings of the colon revealed a violaceous appearance of the mucosa near the ileocecal valve. The mucosa was punctuated by several dark bluish-red plaques. They ranged from 0.3 to about 1.3 cm at their greatest dimension. Some small polyps measuring up to 0.6 cm were also found.

The microscopic picture revealed amyloid involving small blood vessels in the ileum. Significant angiodysplasia of the colon was seen. Dilated submucosal veins were found, and there were areas of mucosal blood vessel ectasia.

Discussion

Yood *et al.*¹ reported a study of 100 patients with amyloidosis that included 18 cases with gastrointestinal tract bleeding, and others with bleeding from other sites. Severe hemorrhage was the cause of death in three patients. They concluded that hemorrhage in amyloidosis was most often due to amyloid infiltration of blood vessels. They reported one case with an episode of epistaxis that required cauterization. Later the patient developed a large retroperitoneal hematoma, which was discovered at surgery. The patient died two weeks later following numer-

* From the Surgical Service, St. Joseph Medical Center, Wichita.

Address reprint requests to W. G. Cauble, M.D., 1148 So. Hillside, Wichita KS 67211.

ous transfusions and an attempt to correct his coagulation abnormalities. In their second reported case, the patient experienced bleeding from an area in the rectum which on biopsy was positive for amyloid. Her liver was enlarged, and a percutaneous biopsy was positive for amyloid.

Levy *et al.*² reported a case in which there was a history of episodes of melena. A "blind" subtotal gastrectomy was done, but the patient continued to have episodes of bleeding. An exploratory laparotomy and other tests failed to demonstrate a source of bleeding. Later x-rays revealed bleeding from the efferent loop of the gastrojejunostomy, and subsequent biopsies taken through the endoscope revealed massive amyloid infiltration. The patient died following a massive episode of melena complicated by cardiac arrest.

These authors feel that bleeding can occur from an ulcer in a markedly infiltrated region. Amyloid infiltration of the musculature can lead to subsequent vascular fragility. The blood supply to a bowel area may become occluded and lead to intestinal ischemia, infarction, or perforation. Portal hypertension due to liver infiltration may cause bleeding from esophageal varices. X-rays of the bowel may reveal a delayed transit time, dilation, or diffuse thickening of the wall. Sometimes small amyloid tumors are seen and these findings may be confused with Crohn's disease or ulcerative colitis.

Johnson *et al.*³ reported a case with a Hemocult positive stool. Gastroduodenoscopy revealed the antrum, pylorus, and duodenal bulb to be very friable but with no distinct ulcers. Biopsies from all three areas revealed amyloid, and the patient was found to have multiple myeloma. He had marked pain, and a barium enema revealed a mass lesion in the descending colon, which was thought to be malignant.

Biopsies revealed amyloidosis. Pandarinath *et al.*⁴ reported a case with abdominal pain in which an upper gastrointestinal series revealed numerous nodular defects throughout the small bowel. A small segment of the jejunum was removed, and it showed massive infiltration throughout the thickness.

Samitz,⁵ in discussing gastrointestinal emergencies, states that the gastrointestinal tract may be affected either locally or diffusely from mouth to rectum with amyloid deposited in and about the walls of small blood vessels, in the fibers of the muscularis mucosae, and in the main casts. Skin and mucous membrane lesions may be characteristic of the disease. These may need to be biopsied.

Summary

Bleeding caused by amyloidosis is rare, and seldom found in a general surgical practice. This case was complicated with amyloidosis of the kidneys and prostate, and the patient was anuric and on dialysis. There have been many studies of the coagulation and bleeding mechanisms, but it has been shown that hemorrhage in amyloidosis is most often due to amyloid infiltration of the blood vessels.

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4. Pandarinath GS *et al.*: Selective massive amyloidosis of the small intestine mimicking multiple tumors. *Radiology* 131:143-146, 1978.
5. Samitz MH: Skin clues to diagnosis of gastrointestinal bleeding and perforation in primary amyloidosis. *Gastrointestinal Emergencies* (Clearfield HR and Dinoso VP Jr., eds.). New York, Grune & Stratton, 1976.

Letters to VOX DOX should be addressed to the Vox Dox Editor, Journal of the Kansas Medical Society, 1300 Topeka Avenue, Topeka, Kansas 66612.

Candidiasis

M. A. LAUVER, M.D. and PATSY BARKER, M.D., *Wichita*

SYSTEMIC fungal infections are of increasing importance in the immunocompromised individual. These generally occur several days to a week after initiation of antibiotic treatment for a suspected bacterial infection. Although *Candida albicans* is most frequently reported, *Candida tropicalis* is increasing in incidence in immune deficient adults. In a recent report, nine of 109 cases (8%) of systemic candidiasis in children were due to *Candida tropicalis*.¹ Presented here are two cases of *Candida tropicalis* septicemia (CTS) in children. One had pericardial involvement, the first such reported case diagnosed antemortem.

Case One

A 12-month-old white male was admitted to St. Francis Regional Medical Center Burn Unit (Wichita) with a 54 per cent partial thickness burn involving the left arm, left leg, anterior right leg, 30 per cent of the anterior trunk, and 9 per cent of the back. The burn was caused by an exploding van radiator.

He was treated with intravenous fluids, salt-poor albumin, and Silvadene dressings. Oral intake was poor. His condition was stable until day nine when he became edematous, lethargic, was intermittently unresponsive, and developed shallow respirations and watery diarrhea. Weight had increased 1.8 kg since admission. He was hyponatremic, hypoalbuminemic, and hypocalcemic. A central venous line was placed in the left external jugular vein, and albumin and calcium were given.

Two days later he had shown no improvement; cultures of blood, urine, wounds, throat, and stool were obtained. *Candida albicans* grew in cultures from the throat and rectum, *Staph aureus* from the throat and urethral discharge; and *Strep liquefaciens* grew from catheterized urine and the wounds. He was begun on cephalothin, nystatin and ampicillin, and repeat cultures were obtained. The urine culture on day 14 grew *Candida tropicalis* in a scant growth. Blood culture had no growth. On day 17, blood culture and jugular catheter tip grew *Candida tropicalis*. Wound culture on day 19 grew *Candida tropicalis* and *Candida albicans*. Amphotericin was begun at 0.25 mg/kg advancing to 1 mg/kg in 0.25 mg/kg daily increments with ketoconazole, 50 mg/day orally. The mean inhibitory concentration of amphotericin for this *Candida tropicalis* was 0.39 µg/ml, and the mean fungicidal titer was 0.78 µg/

ml. Peak levels of amphotericin were 1.1 µg/ml and trough 0.4 µg/ml. No titers or levels could be determined with the ketoconazole. Creatinine was 0.4 mg/dL at the beginning of therapy. On day 20, liver and heart were enlarged and chest x-ray showed perihilar infiltrates and fluid. Echocardiogram was negative for pericardial effusion. He was begun on digoxin. Ampicillin was discontinued after 20 days, and he began to show steady improvement. *Candida tropicalis* grew in blood five days after amphotericin was begun and in urine six days later. The right internal jugular line was discontinued on the 27th hospital day as was the cephalothin, nine days after amphotericin was begun. After 12 days of daily therapy, amphotericin was changed to alternate days. After 13 days of amphotericin, the blood culture showed no growth. Ketoconazole was discontinued after 14 days and amphotericin after 12 days of daily therapy and seven days of alternate day therapy. Total dose of amphotericin was fifteen mg/kg. Blood and urine cultures on the day therapy was discontinued were negative. He was discharged on the 38th day in good condition.

Case Two

A 4-year-old white female was admitted with a one-month history of fever, strep throat, anorexia, malaise, shortness of breath, and lethargy. Bone marrow aspiration showed acute lymphocytic leukemia-null cell type. She was neutropenic throughout her hospitalization.

She was febrile and tobramycin, nafcillin, and ticarcillin were started. She became afebrile, bacterial cultures were negative, and antibiotics were discontinued on day nine. On the 19th hospital day she again became febrile, and triple antibiotic therapy was resumed. *E. coli* grew in the blood culture. An ecchymotic lesion was present on the right upper arm. Fungal and bacterial cultures of the lesion were negative. *Candida tropicalis* grew in the blood culture on the 21st hospital day. Ketoconazole was begun on the 23rd day, and amphotericin was added on day 26. At the start of therapy, creatinine was 0.5 mg/dL, and blood urea nitrogen 6 mg/dL. On the 26th day the heart size was enlarged and sounds muffled. Pericardial fluid was shown by echocardiogram.

A pericardial tap grew *Candida tropicalis*. Seven days after amphotericin was begun, the blood cul-

ture was negative but *Candida tropicalis* and *Candida albicans* grew in cultures from the throat and urine. On day 37, after 12 days of amphotericin, the culture from a repeat pericardial tap grew *Candida tropicalis*. Blood culture at that time also grew *Strep viridans*.

On the 39th hospital day she developed diarrhea and agitation, and on the 40th day she suffered a cardiopulmonary arrest. She was resuscitated and placed on the respirator. On the 42nd day the EEG was flat, and the parents asked that the respirator be discontinued. No autopsy was performed.

Discussion

The incidence of *Candida tropicalis* is variously reported as 20-30 per cent of systemic fungal infections with one report of 80 per cent.² Of those with urinary or respiratory tract colonization by *Candida tropicalis*, 80 per cent develop systemic disease whereas only 7 per cent with *Candida albicans* colonization do so.³ Most reports of septicemia are associated with neutropenia, chemotherapy and antibiotics, or with burns treated by antibiotics.⁴

Mechanical and immunologic factors contribute to susceptibility to systemic candidiasis. Bacteria that occupy the body surfaces produce antifungal substances, and intact skin is an inadequate medium for growth of *Candida*. *Candida* induces a specific cell mediated immunity, and a deficiency of the cellular immune system is associated with increased susceptibility to systemic candidiasis. Phagocytosis by neutrophils, monocytes, and eosinophils is a major defense mechanism.⁵ Other risk factors for the development of systemic fungal infections include total parenteral nutrition which is rich in nitrogen, an ideal growth medium; the presence of an intravenous catheter;^{6, 7} irritation of the cannulated vein by intravenous solutions; malnutrition and decreased protein synthesis which leads to decreased immunoglobulin production; and the use of broad spectrum antibiotics causing destruction of competitive normal flora.

Candida species are often considered part of the normal flora of body surfaces or fluids, but outside their "natural" habitat become pathogens. Children who develop systemic candidiasis have wound surface cultures and urine cultures positive for *Candida* prior to developing candidemia.⁸

The first case presented is a burned child who was never neutropenic, but who had received broad spectrum antibiotics and oral nystatin for colonization with *Candida albicans*. *Candida tropicalis* is less sensitive to nystatin and this may have contributed to selection of this organism. He also had a central

venous catheter in place. Blood cultures remained positive for *Candida tropicalis* as long as the central line was in place in spite of amphotericin and ketoconazole therapy. Sensitivity of this particular organism to ketoconazole is unknown. The reported in vitro mean inhibitory concentrations of ketoconazole for *C. tropicalis* range from 0.1 µg/ml to 64 µg/ml.⁹ Peak levels of amphotericin in this patient were three times the mean inhibitory concentration and 1.5 times the mean fungicidal titer. During therapy the burns were healing well and the immunologic system was returning to normal.

The second case is a child with acute lymphocytic leukemia who was neutropenic and had received both chemotherapy and broad spectrum antibiotics. In systemic disease *Candida* species frequently invade the heart producing myocarditis or endocarditis. However, only eight cases of purulent pericarditis due to *C. tropicalis* have been reported. It was fatal in 100 per cent of the cases.^{10, 11} This is the first known case in a child in which *C. tropicalis* pericarditis has been identified antemortum. Despite therapy for 12 days with amphotericin B and 15 days with ketoconazole, the organism persisted in the pericardial fluid. Since no autopsy was performed, it is unknown if the pericardial fluid was clear after another five days of therapy, or if myocarditis or endocarditis contributed to the patient's death. *Strep viridans* was present in a blood culture at the time of death.

The recommended therapy for systemic candidiasis is amphotericin B, beginning with 0.25 mg/kg per day and progressing to 1 mg/kg per day at 0.25 mg/kg intervals. Optimal duration of therapy is unknown and may vary for each organism or host. A total dose of 15-45 mg/kg during the course of therapy has been recommended.¹²

Synergism of amphotericin with flucytosine or miconazole has been noted in vivo.¹³ In our burn patient, the *Candida tropicalis* was not sensitive to either of these drugs. Ketoconazole is an imidazole drug (as is miconazole), and the efficacy of this in combination with amphotericin in patients with *Candida tropicalis* septicemia is unknown. Both of our patients were treated with amphotericin and ketoconazole. In vitro studies with this combination in treating *Candida albicans* have variously shown synergism or antagonism. Brajtburg reported that the in vitro effect of short term incubation of this combination with *Candida albicans* was neither synergistic nor antagonistic; however long term incubation produced synergism.¹⁴ There is some evidence that an imidazole drug can inhibit amphotericin binding to

(Continued on page 297)

Differential Lung Ventilation

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DIFFERENTIAL lung ventilation (DLV) is the selective ventilation of each lung by means of two ventilators connected to the outlets of a double-lumen endobronchial tube. If properly positioned in the upper airway, different ventilatory parameters can be provided for each lung. Although this technique has long been employed for anesthetic administration in thoracic surgery, the continuance of endobronchial intubation has only recently been utilized for extended periods. The development of double-lumen tubes with low pressure cuffs has reduced the morbidity of prolonged intubation, and has permitted the selective delivery of positive pressure to each hemithorax. The case report illustrates the effective employment of DLV following lung decortication complicated by continuing hemorrhage and massive air leak.

Case Summary

A 67-year-old white male was admitted to the hospital complaining of right sided back pain and increasing lethargy. He had a two year history of a myeloproliferative disorder which had made him transfusion dependent. He was taking prednisone, 40 mg per day. The rest of his health history was essentially noncontributory; he had smoked cigarettes for 40 pack-years, but had no pulmonary diseases prior to this admission.

Chest x-ray revealed a right pleural effusion, which was drained. Subsequently he developed bacteremia with spiking elevations of temperature. He was transferred to the Bell Memorial Hospital, where on admission his temperature was 38C; arterial blood gases (ABGs) were pH 7.56, PaO₂ 52 torr, and PaCO₂ 39 torr. The chest roentgenogram showed fluid in the dependent portion of the right chest and patchy upper lobe densities. Thoracentesis yielded pus.

The following day a right thoracotomy was performed to establish drainage. Multiple small ab-

cesses were found on the surface of the lung. When these were excised, bleeding became difficult to control, and multiple air leaks developed. Endotracheal ventilation became increasingly inefficient. When bleeding made it necessary to return the patient to the operating room a few hours later, an endobronchial tube (Broncho-Cath, National Catheter Co.) was introduced. The patient had received a total of 20 units of blood, and adequate hemostasis was achieved with difficulty. A decision was made to employ DLV postoperatively.

Two ventilators (Servo Ventilator 900C, Siemens-Elcoma) were used, with one controlling the time of inspiration of the other (master and slave) via a connecting cable. Thus the two machines were synchronized to begin inspiration simultaneously. Ventilation of both lungs was controlled. The master ventilator was connected to the intact (left) lung, and was set to function as a constant flow generator, with a tidal volume of 500 ml, and with positive end-expiratory pressure (PEEP) of 10 cmH₂O. The slave ventilator was connected to the right lung, and was set to function as a pressure generator with a limit of 20 cmH₂O. Five cmH₂O of PEEP was applied to this lung. The respiratory rate was gradually reduced from 16 to 12 breaths per minute. At an FIO₂ of 0.5 the ABGs were pH 7.53, PaO₂ 133, PaCO₂ 41.

Bleeding from the right chest virtually ceased within two hours after DLV was started. Vital signs remained stable; the cardiac output was 8 liters per minute. Representative ventilatory measurements taken on the second day of DLV are shown in *Table I*. The air leak gradually disappeared. On the fourth day the FIO₂ was decreased to 0.4, and intermittent mandatory ventilation was started with a mandatory rate of eight breaths per minute. Chest x-rays showed the right lung to be well expanded. Six days after DLV was instituted the patient was extubated and released from the intensive care unit. He was discharged from the hospital five weeks postoperatively.

Discussion

Ventilating the lungs individually has many possible applications, since lung diseases and injuries are often unilateral. The first double-lumen endobron-

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TABLE I
MEASUREMENTS: DLV DAY #2

	Left Lung (Intact)	Right Lung (Air Leak)
Inspired tidal volume (ml)	504	325
Expired tidal volume (ml)	625	250
Expired minute volume (liters per min)	4.3	2.6
Peak inspiratory pressure (cmH ₂ O)	24.3	15.0
Pause pressure (cmH ₂ O)	17.0	12.5
Mean airway pressure (cmH ₂ O)	7.8	3.6

chial tube — the Carlens tube — was introduced for the surgical management of patients with bronchiectasis or lung abscess, the primary indication being to prevent cross-contamination of the intact lung by the infected one.¹ Subsequently, one-lung anesthesia has become widely employed for lateral thoracotomy, where better surgical exposure can be effected by collapsing the lung on the operated side. The Carlens tube has since been superseded by better models (Bryce-Smith,^{2, 3} White,⁴ Robertshaw⁵). The subject of endobronchial tube design has recently been reviewed.⁶

If lung pathology is essentially unilateral, alveolar ventilation and diffusion can be apportioned between the lungs according to individual circumstances. Probably the commonest condition where DLV can be used to advantage is unilateral atelectasis or pneumonia. Conventional endotracheal ventilation tends to distribute the inspired air preferentially to the more compliant lung, favoring the process of consolidation in the diseased lung. This causes increased ventilation-perfusion mismatching, venous admixture, and hypoxemia. DLV has been shown in several studies to permit adjustments of positive pressure to each lung, so that reexpansion of the affected lung is accomplished, ventilation and perfusion are better balanced, and the PaO₂ is raised toward a normal value.⁷⁻⁹

Less common, but often more acutely serious, is the lung with a large air leak, as seen in this instance

after decortication, or with a bronchopleural fistula. Here resistance to inspiratory airflow is *less* on the affected side. If thoracostomy drainage is instituted, endotracheal positive pressure will drive much of the inspired air through the leak and out the chest tube. In the past some patients have been benefited by ventilating only the intact lung via an endobronchial tube. However, the resulting collapse of the leaking lung is almost invariably followed by pneumonia. DLV offers a therapeutic approach to the problem by allowing only enough positive pressure to be delivered to the leaking lung to prevent its collapse. The intact lung can usually provide most of the patient's necessary gas exchange.

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Candidiasis

(Continued from page 295)

the fungal cell membrane through inhibition of ergosterol synthesis.¹³ Until in vivo studies determine whether ketoconazole and amphotericin combination therapy is synergistic or antagonistic, routine use cannot be recommended.

References are available from Dr. Lauver, St. Francis Regional Medical Center, 929 No. St. Francis, Wichita KS 67214.

Multiple Leiomyomata of the Urinary Bladder

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EPITHELIAL tumors of the urinary bladder are common lesions and have received much attention. Malignant tumors of muscle comprise the second most frequent group of tumors of the bladder after epithelial tumors. However, benign mesothelial tumors of the bladder are rare. Leiomyomas of the bladder constitute a relatively small percentage of this rare group and should be considered in the differential diagnosis of bladder masses.

In 1953, Campbell and Gislason¹ reported a case of leiomyoma of the urinary bladder, reviewed the literature, and reported a total of 68 cases. Since then, knowledge of leiomyomas of the bladder has been based on a few single case reports because few urologists have had the opportunity for wide experience with these lesions. We report here a patient with a submucosal leiomyoma of the bladder neck, treated by transurethral resection, who subsequently developed a second leiomyoma.

Case Report

A 63-year-old white male was admitted to the Veterans Administration Medical Center, Leavenworth, because of acute urinary retention after two weeks of weak urinary stream and dysuria. He had previously undergone a transpubic radical prostatectomy because of stage B1 adenocarcinoma of the prostate gland and urethral diverticulectomy. Physical examination was unremarkable. Laboratory studies yielded results that were within normal limits.

Cytoscopic examination revealed a polypoid formation at the level of the bladder neck. The patient underwent a transurethral resection, and multiple fragments of brownish-gray to brownish-white, glistening, rubbery tissue were removed. These were of assorted shapes and varied in size from 0.3 to 0.9 cm. Microscopic examination showed a submucosal leiomyoma of the urinary bladder (*Figure 1*).

Six months later, while the patient was asymptomatic, a cytoscopic examination revealed a small polypoid lesion at the anterior wall of the bladder, which could not be reached with the resectoscope.

The patient underwent partial cystectomy, and a 1.3 cm smooth-walled, polypoid lesion was excised from the right anterior wall of the bladder (*Figure 2*). Microscopic examination showed a submucosal leiomyoma of the urinary bladder (*Figure 3*). His postoperative course was unremarkable.

Comment

Leiomyomas may appear at any site in the urogenital system. They have been reported in the kidney, bladder, urethra, penis, epididymis, prostate, scrotum, seminal vesicles, and spermatic cord. The kidney is probably the most frequent site involved.² Leiomyomas of the bladder occur in all age groups and have been reported from the first to the eighth decade.³ It appears that leiomyomas are three times more common in female than in male patients.² Leiomyomas can be classified by location as subserosal, intramural, or submucosal.⁴ Smaller lesions usually are sessile. Later the tumors enlarge to become pedunculated and may protrude through the bladder neck. Subserosal leiomyomas can become very large and present with symptoms secondary to the mass. Leiomyomas of the bladder are usually found to involve the trigone, and symptoms are directly related to the location and size of the tumor.⁵ The patient may present with urinary tract infection, obstruction, hematuria, suprapubic or perineal pain, or — in females — prolapse of the lesion through the urethral meatus. Grossly, leiomyomas are firm, rubbery, gray-white masses,

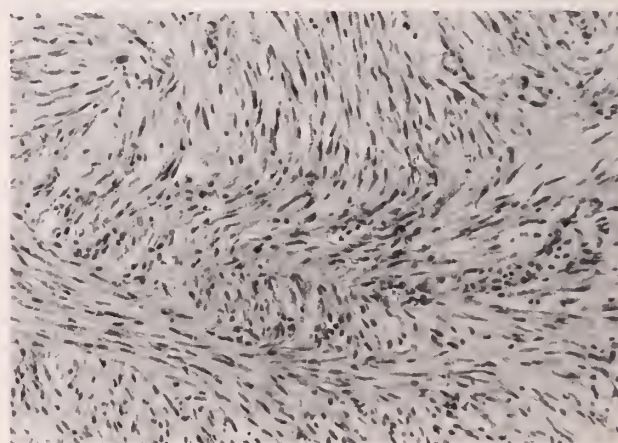


Figure 1. Interlacing bundles of smooth muscle fibers.

* Formerly of The Veterans Administration Center, Pathology and Urology Services, Leavenworth.

Address reprint requests to Dr. Chavez, the Moser Clinic, P.A., 418 West 5th Street, Holton KS 66436.



Figure 2. Cut section of polypoid lesions from bladder.

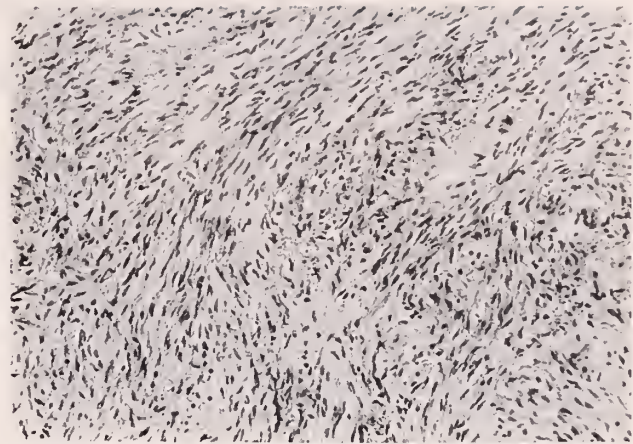


Figure 3. Bundles of smooth muscle fibers showing tendency to whorl type of arrangement.

usually 3 to 5 cm in diameter. Microscopically, these tumors are composed of diffuse interlacing bundles or whorls of smooth muscle fibers. Radiographically, submucosal and intramural leiomyomas of the bladder usually present as smooth, filling defects on cystogram, but may be difficult to differentiate from other bladder tumors.⁶ To date there have been no reports of malignant degeneration.² Local but complete excision by open surgery or transurethral resection are the treatments of choice.

It appears that our patient represents the first reported case of multiple leiomyomata of the urinary bladder.

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A Change in the Air

There are different ways to be born again, and the Editorial Board has plans to introduce its readers to some with the January issue of the JOURNAL OF THE KANSAS MEDICAL SOCIETY.

Although the JOURNAL has long since passed its infancy, it may well suggest a new-born in the number of changes apparent.

The Board does submit, however, that there was nothing natural about this rebirth.

The President's Message

The tally is in. Two-thirds of Kansas physicians signed with Medicare as participating physicians. The exact numbers are 1457 of 2166 MDs in all of Kansas (excluding Wyandotte and Johnson counties which are in another Medicare region) — for 67 per cent participation.

The national average is less than 20 per cent. Questions have been asked: "Why does Kansas lead the nation in percentage of Medicare participating physicians? Why is the percentage in Kansas double to more than triple the percentages in surrounding states? What are the factors that have influenced the vast majority of Kansas physicians to elect to be bound tightly by national health insurance in caring for a large segment of the population?"

One must realize the history of the cooperation of the sunflower physicians with the Blues. In 1983, 87.5 per cent of all Kansas physicians signed participating contracts with Blue Shield. This figure rose to 90.5 per cent in 1984. The first of this year, CAP was implemented. On January 1, 1984, Kansas Blue Cross became the first non-governmental insurance program to use DRGs for hospital payment. The Kansas physician has worked in and with these programs and understands them. In March 1984, the Council of the Kansas Medical Society followed the lead of the AMA Board of Trustees and voted overwhelmingly to recommend a voluntary freeze of all fees by members of the KMS for a 12-month period. The physicians of Kansas have historically responded to requests from senior citizens for consideration of people on fixed incomes. There is a high rate of acceptance of assignment on Medicare claims even in the group of Kansas physicians who are philosophically opposed to signing a Medicare participating physician's agreement.

Times change. And people change with time. Who would have thought 50 years ago that Kansas would be one of the leaders in medical collectivism?

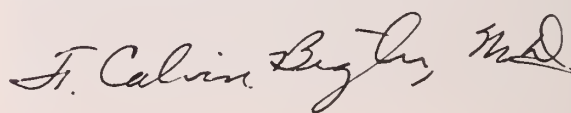
There was a time when the physician and the patient and/or the family of the patient met together and agreed upon the fee for the care of an illness. The monetary value of the professional service to that particular patient was a definite factor considered in setting the fee. Thus, the fee might be quite different for the same operation depending upon whether that

operation was done on an elderly widow or an active young businessman. There often was a correlation in the size of the fee to the increase or preservation of earning power of the patient.

Times have changed. Physicians are locked into fee schedules that are relatively rigid. A physician is working in an increasingly regulated climate. S/he is increasingly regulated as to fees, requirements in medical education, licensure, hospital privileges, business practices. There is more and more regulation in everything in a physician's life — with one exception.

The one thing that has gone totally unregulated is that physician's exposure to the hazard of professional liability. It is inconsistent with the overall picture for a physician to be held liable for the entire earning power of a malpractice litigant when in taking care of that patient the physician is not compensated by a fee related to the saving and increasing of that patient's earning power. A society that imposes arbitrary controls on earnings of its healing profession has a similar right — and duty — to protect those healers from being destroyed by an uncontrolled system of payment for mistakes. Thus, there certainly is justification for a reasonable maximal limit on a professional liability award — even though that limit may well be far below potential economic damages. And there is a need to ensure that the damaged patient receives the bulk of the compensation by placing the attorney in the same societal milieu of professional control as the physician.

Whether or not our efforts will be found to be historically commendable, we in Kansas are leading the way into increasing medical collectivism. Kansas must also lead in solving the malpractice crisis. Professional liability must be rationally regulated or the stresses produced by the anachronism between the systems governing medical practice versus the systems governing malpractice will erode and eventually destroy health care as we know it.



President



The Cherished Burden

Anyone scanning medical publications during the past few years is certain to notice a change in content. Material relating to the social, economic, and political factors of medical practice has encroached increasingly on the professional and technical subjects. Indeed, some journals, state and regional in particular, have gone the full measure and given themselves over completely to such matters.

Again, anyone surveying the medical literature would be certain that physicians themselves are afflicted with excessive states of rubor and calor not to mention large dollops of dolor. There is, it would seem, less and less of the ecstasy and more and more of the agony to be encountered in practice these days. This is not an entirely balanced interpretation — understandable perhaps because of the excessive dosage but obscuring the fact that there have been no significant defections from the medical ranks. True, there has been much soul-searching and a few early retirements are reported, but on balance, no apparent decrease in novices seeking admission to the order.

Still, in the quieter moments between tussling with the socioeconomic factors and professional obligations, there must be some degree of reflection on how this unsettled state of being has evolved. Behind the more apparent factors of social alteration, political demands, and economic realities, not to mention medicine's intrinsic revolution, there must be some compulsion affecting the medical world in particular, and we offer for contemplation the proposition — or rather the question: are we going too fast? Are these problems a reflection that the medical fare is being prepared and served too fast for the social digestive system to assimilate properly?

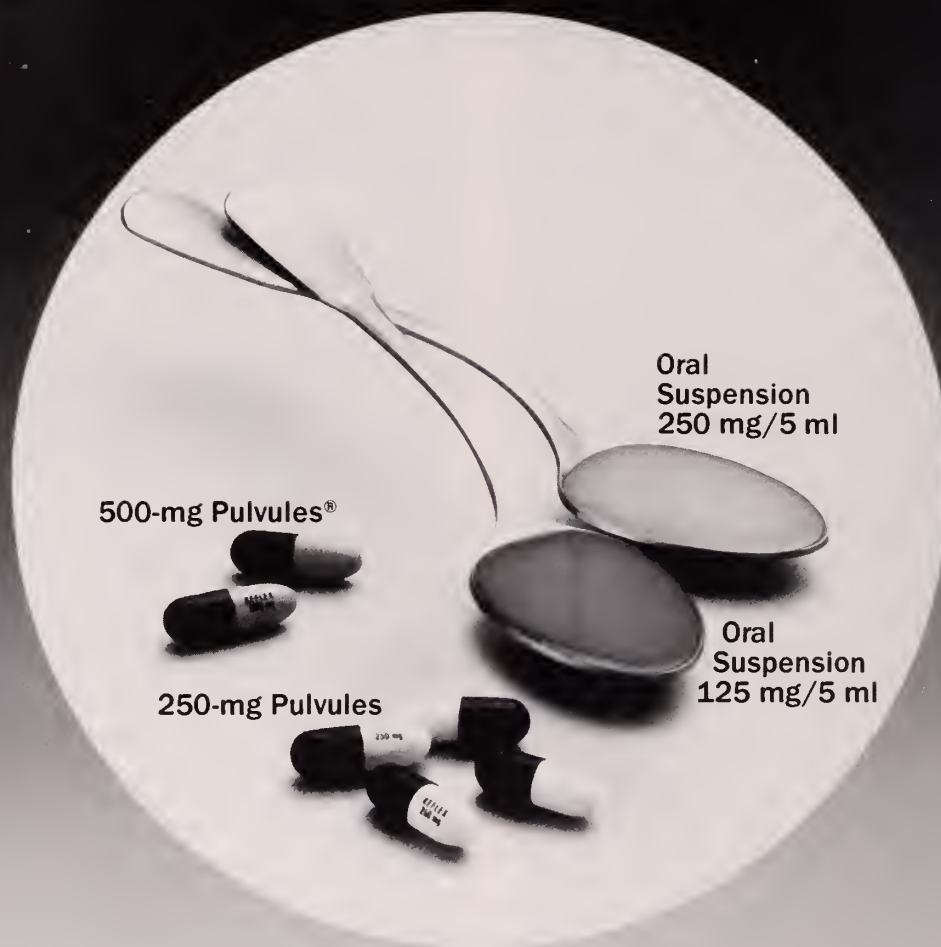
It is, of course, one of those questions anyone can develop an answer to and no one *the* answer. A case can certainly be made, however, for the proposition that, insofar as medical methodology and personnel and capability have affected the social patterns and been affected by them, a highly inefficient, even extravagant, system has evolved — if, that is, one looks only at the obvious and immediately measurable economic features. We can point to lives that

have been saved, in itself an uncertainty: while we can say that life expectancies have been extended, how does one put that in personal terms and say that this or that person represents one of the clearcut dividends of the system? Moreover, the speed of our therapeutic efforts keeps, on the forward edge, a number of survivors who are not directly productive by their survival (except, by their continued presence, to themselves and their families). To medicine, they are a limited victory. To society, they are a burden since the accomplishment of this survival is, by its novelty, expensive, and there remains the necessity to support them. Does society see them as an investment in the future since, from them, we learn new methods that *will* later sustain some (and increase our gross national product)?

But this isn't a fixed scenario. In fact, it is difficult to develop one because the scene changes too rapidly. It is this complex of conflicts — the public demand for optimum care including the latest (if untried) developments against the profession's need to evaluate properly, control methods, develop personnel to supply it — and justify the cost. This contributes to the liability problem because it exposes the profession to the public's expectation of an unattainable perfection of performance. It can become a prime factor in separating patient from physician since the availability of such services frequently calls for the introduction of other groups of individuals unfamiliar to the patient (and therefore unloved) even as the patient may be seen by those groups primarily as another episode in their accumulated experience by which they expect to develop perfection and justify procedures. Fortunately, there is a firm bedrock of less esoteric medical service which binds patient and physician in the more commonplace efforts, and that is what maintains the laudable image of the physician.

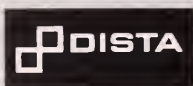
Where do we stop? Of course, we don't. Are we going too fast? Not unless someone can come up with an answer to that eternal question, what is the measure of human life in fiscal terms? Only then can an economically dictated limit be placed on our efforts, both of the moment to sustain and of the future in the search for new ways. However grim and seemingly destructive to such a spirit the current upheaval may be, the medical mind is incorrigible. It will continue its efforts and present to society the problems of how to deal with medical successes. Society will give its urgent approval to the promise — and rail against the cost. — D.E.G.

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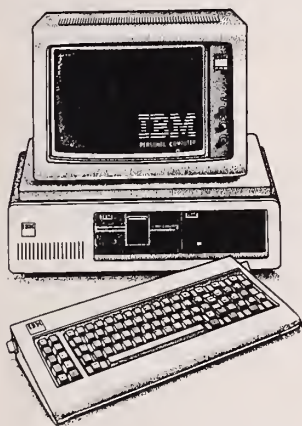
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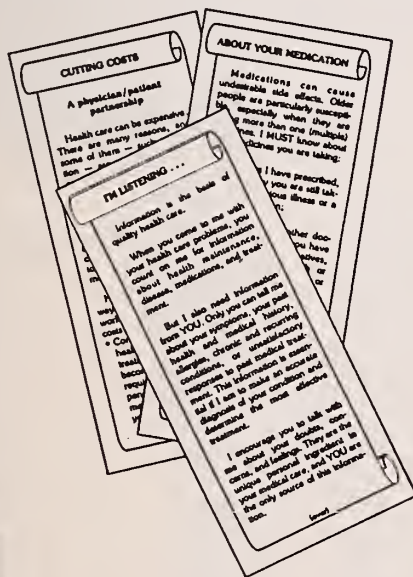
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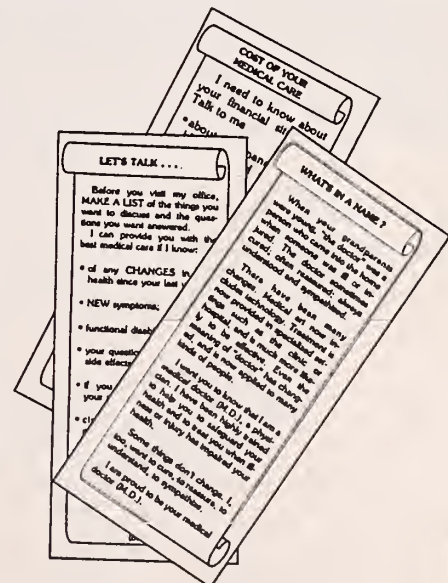
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Precautions: ISOPTIN should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Studies in a small number of patients suggest that concomitant use of ISOPTIN and beta blockers may be beneficial in patients with chronic stable angina. Combined therapy can also have adverse effects on cardiac function. Therefore, until further studies are completed, ISOPTIN should be used alone, if possible. If combined therapy is used, patients should be monitored closely. Combined therapy with ISOPTIN and propranolol should usually be avoided in patients with AV conduction abnormalities and/or depressed left ventricular function or in patients who have also recently received methyl dopa. Chronic ISOPTIN treatment increases serum digoxin levels by 50% to 70% during the first week of therapy, which can result in digitalis toxicity. The digoxin dose should be reduced when ISOPTIN is given, and the patient carefully monitored. ISOPTIN may have an additive hypotensive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after ISOPTIN administration. Until further data are obtained, combined ISOPTIN and quinidine therapy in patients with hypertrophic cardiomyopathy should probably be avoided, since significant hypotension may result. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. **Pregnancy Category C:** There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. It is not known whether verapamil is excreted in breast milk; therefore, nursing should be discontinued during ISOPTIN use.

Adverse Reactions: Hypotension (2.9%), peripheral edema (1.7%), AV block: 3rd degree (0.8%), bradycardia: HR<50/min (1.1%), CHF or pulmonary edema (0.9%), dizziness (3.6%), headache (1.8%), fatigue (1.1%), constipation (6.3%), nausea (1.6%). The following reactions, reported in less than 0.5%, occurred under circumstances where a causal relationship is not certain: confusion, paresthesia, insomnia, somnolence, equilibrium disorders, blurred vision, syncope, muscle cramps, shakiness, claudication, hair loss, maculae, and spotty menstruation. Overall continuation rate of 94.5% in 1,166 patients.

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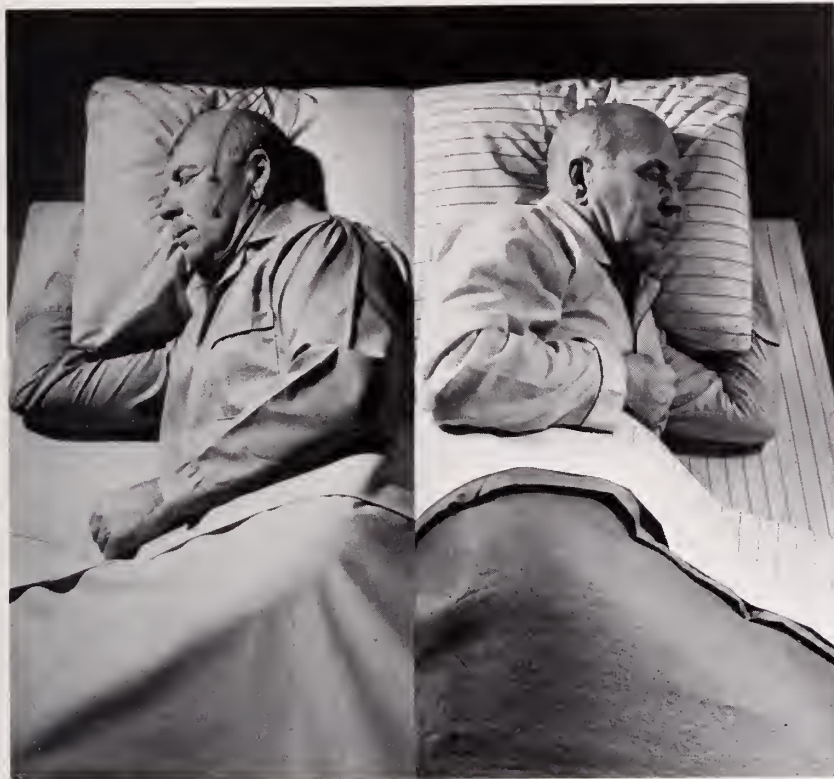
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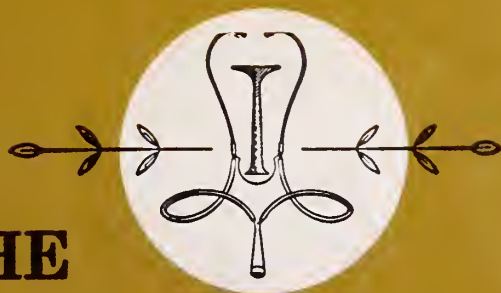
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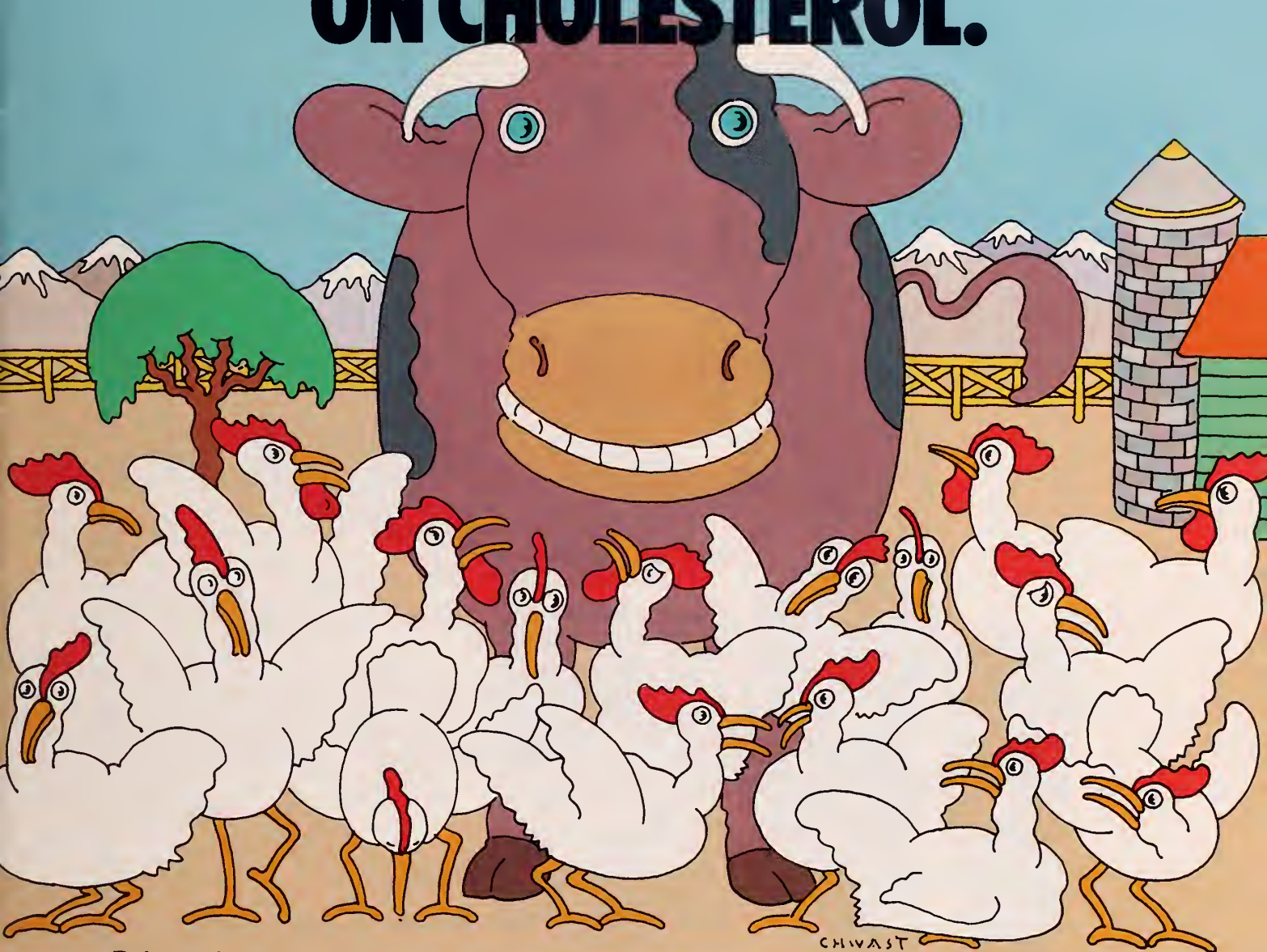
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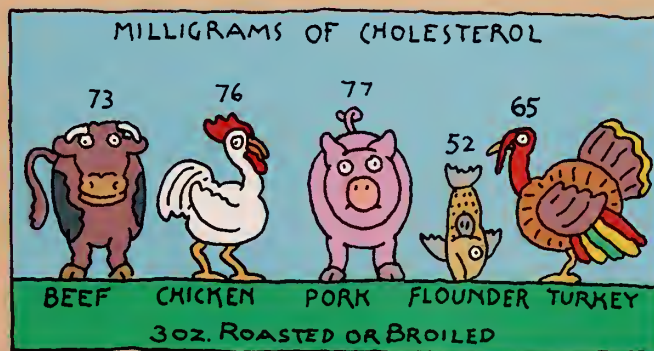
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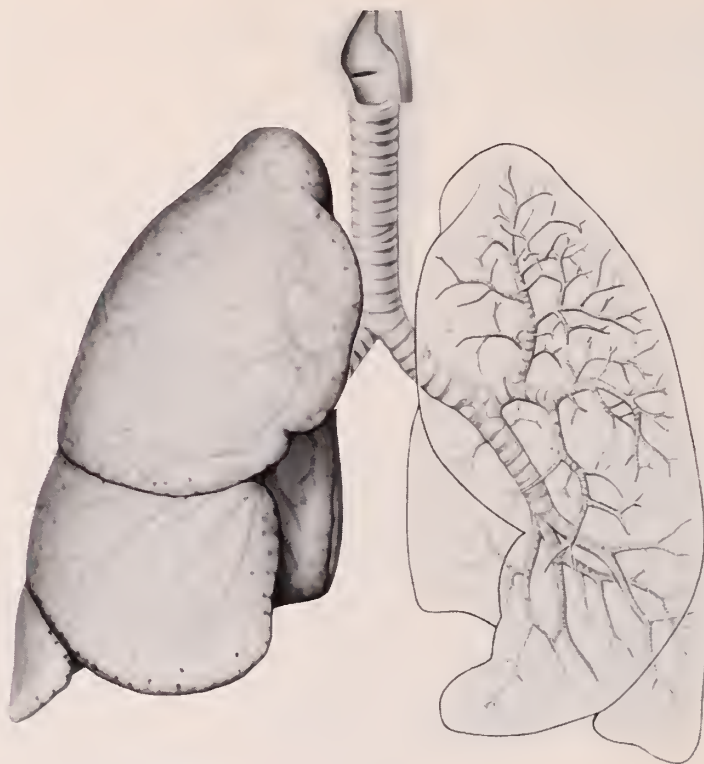


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Indications and Usage Cecilor* (cefactor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

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Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Cecilor.

Contraindication Cecilor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings IN PENICILLIN SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS, INCLUDING ANAPHYLAXIS, TO BOTH DRUG CLASSES.

Antibiotics, including Cecilor, should be administered cautiously to any patient who has demonstrated some form of allergy particularly to drugs.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, manage-

ment should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C. difficile*. Other causes of colitis should be ruled out.

Precautions **General Precautions** — If an allergic reaction to Cecilor* (cefactor, Lilly) occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids. Prolonged use of Cecilor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics in hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition; it should be recognized that a positive Coombs' test may be due to the drug.

Cecilor should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

As a result of administration of Cecilor, a false positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinistest[®] tablets but not with Tes-Tape[®] (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy — **Pregnancy Category B** — Reproduction studies have been performed in mice and rats at doses up to 12 times the human dose and in ferrets given three times the maximum

human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Cecilor* (cefactor, Lilly). There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers — Small amounts of Cecilor have been detected in mother's milk following administration of single 500-mg doses. Average levels were 0.18, 0.20, 0.21, and 0.16 mcg/ml at two, three, four, and five hours respectively. Trace amounts were detected at one hour. The effect on nursing infants is not known. Caution should be exercised when Cecilor is administered to a nursing woman.

Usage in Children — Safety and effectiveness of this product for use in infants less than one month of age have not been established.

Adverse Reactions Adverse effects considered related to therapy with Cecilor are uncommon and are listed below.

Gastrointestinal symptoms occur in about 2.5 percent of patients and include diarrhea (1 in 70).

Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely.

Hypersensitivity reactions have been reported in about 1.5 percent of patients and include morbilliform eruptions (1 in 100), pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of serum-sickness-like reactions (erythema multiforme or the above skin manifestations accompanied by arthritis/arthritis and, frequently, fever) have been reported. These reactions are apparently due to hypersensitivity and have usually occurred during or following a second course of therapy with Cecilor. Such reactions have been reported more frequently in children than in adults. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.

Cases of anaphylaxis have been reported, half of which have

occurred in patients with a history of penicillin allergy. Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain — Transitory abnormalities in clinical laboratory test results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic — Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic — Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

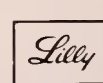
Renal — Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200).

(061782A)

Note. Cecilor* (cefactor, Lilly) is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

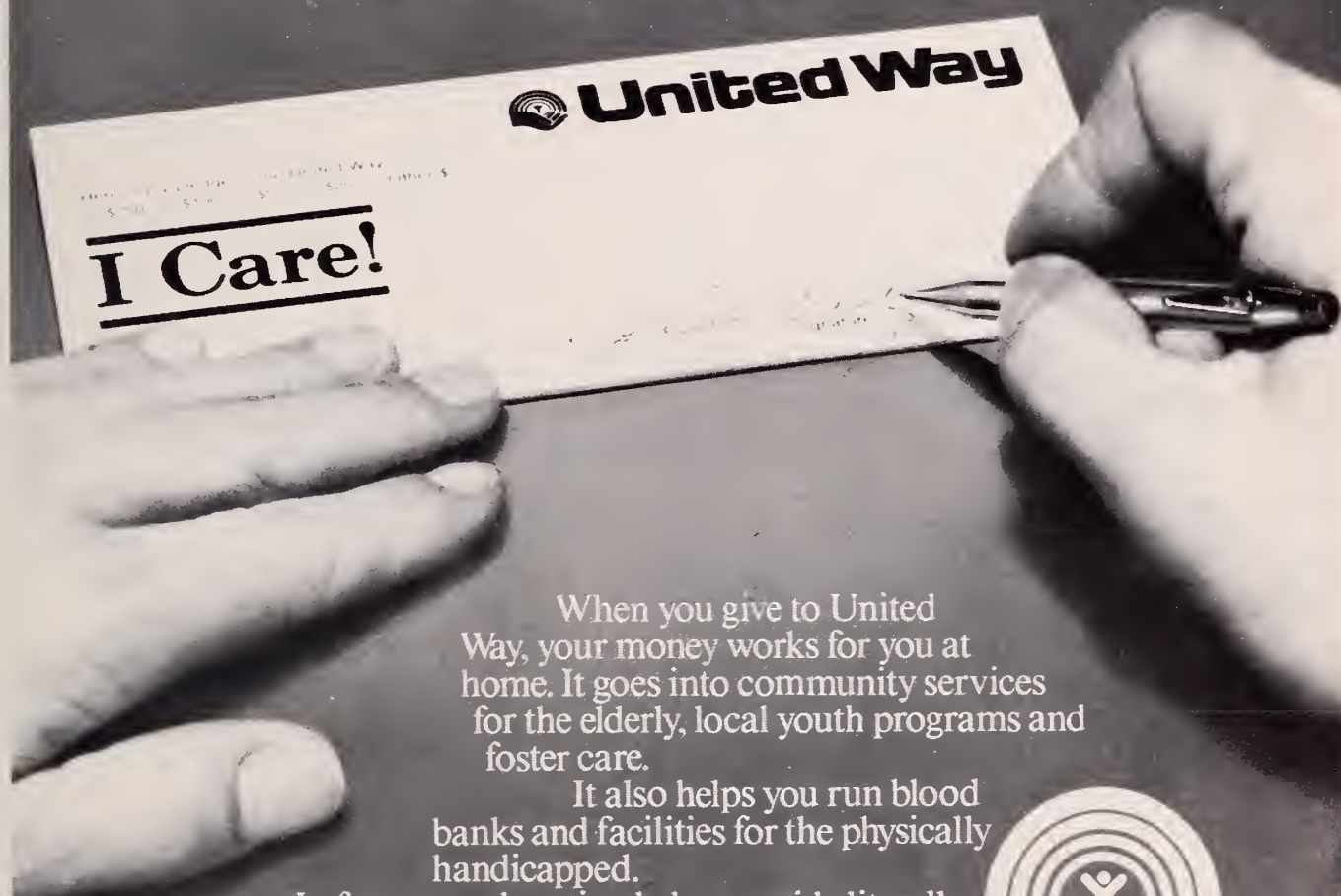
Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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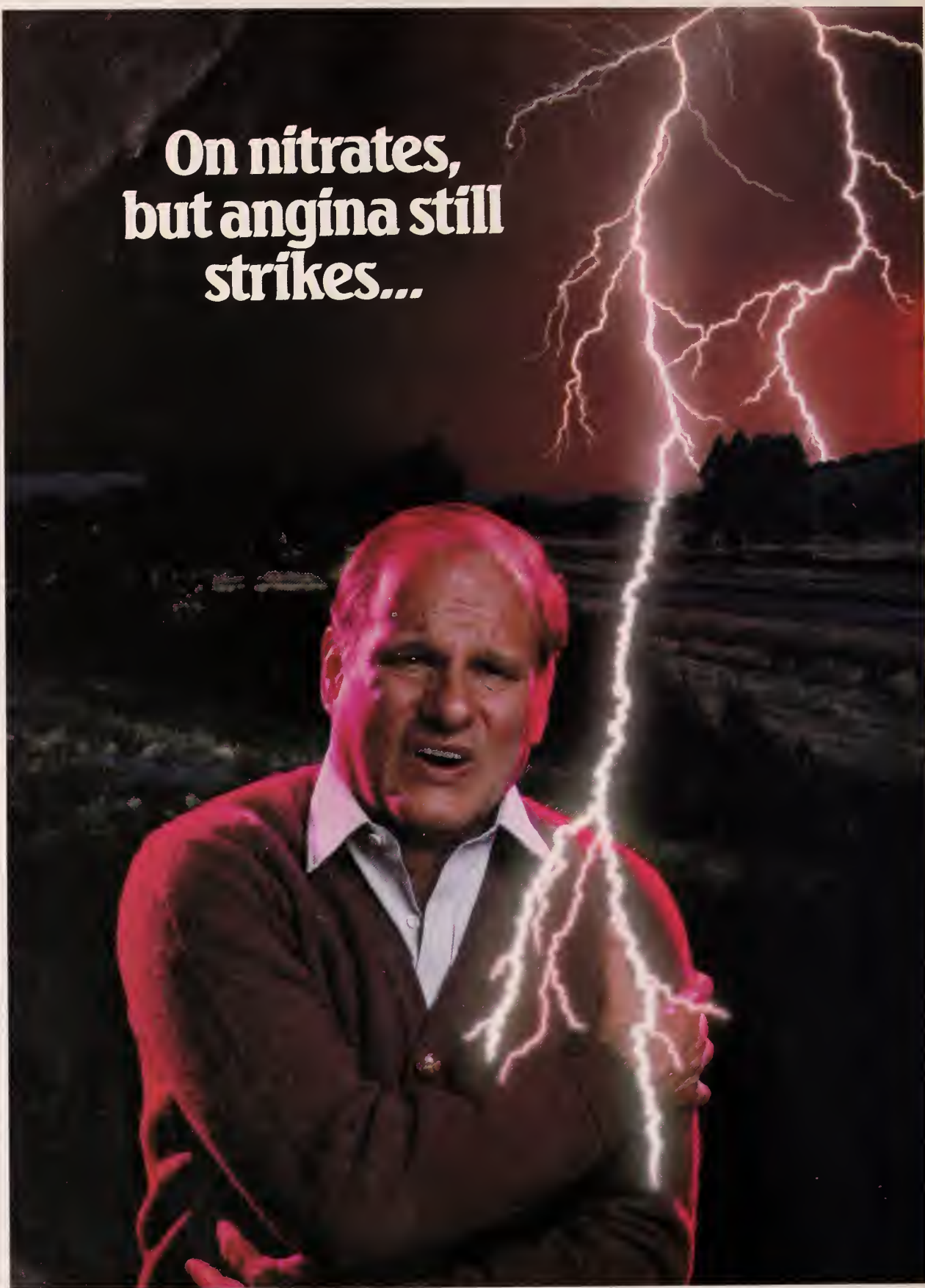
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Precautions: ISOPTIN should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Studies in a small number of patients suggest that concomitant use of ISOPTIN and beta blockers may be beneficial in patients with chronic stable angina. Combined therapy can also have adverse effects on cardiac function. Therefore, until further studies are completed, ISOPTIN should be used alone, if possible. If combined therapy is used, patients should be monitored closely. Combined therapy with ISOPTIN and propranolol should usually be avoided in patients with AV conduction abnormalities and/or depressed left ventricular function or in patients who have also recently received methylidopa. Chronic ISOPTIN treatment increases serum digoxin levels by 50% to 70% during the first week of therapy, which can result in digitalis toxicity. The digoxin dose should be reduced when ISOPTIN is given, and the patient carefully monitored. ISOPTIN may have an additive hypotensive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after ISOPTIN administration. Until further data are obtained, combined ISOPTIN and quinidine therapy in patients with hypertrophic cardiomyopathy should probably be avoided, since significant hypotension may result. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. **Pregnancy Category C:** There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. It is not known whether verapamil is excreted in breast milk; therefore, nursing should be discontinued during ISOPTIN use.

Adverse Reactions: Hypotension (2.9%), peripheral edema (1.7%), AV block: 3rd degree (0.8%), bradycardia. HR<50/min (1.1%), CHF or pulmonary edema (0.9%), dizziness (3.6%), headache (1.8%), fatigue (1.1%), constipation (6.3%), nausea (1.6%). The following reactions, reported in less than 0.5%, occurred under circumstances where a causal relationship is not certain: confusion, paresthesia, insomnia, somnolence, equilibrium disorders, blurred vision, syncope, muscle cramps, shakiness, claudication, hair loss, maculae, and spotty menstruation. Overall continuation rate of 94.5% in 1,166 patients.

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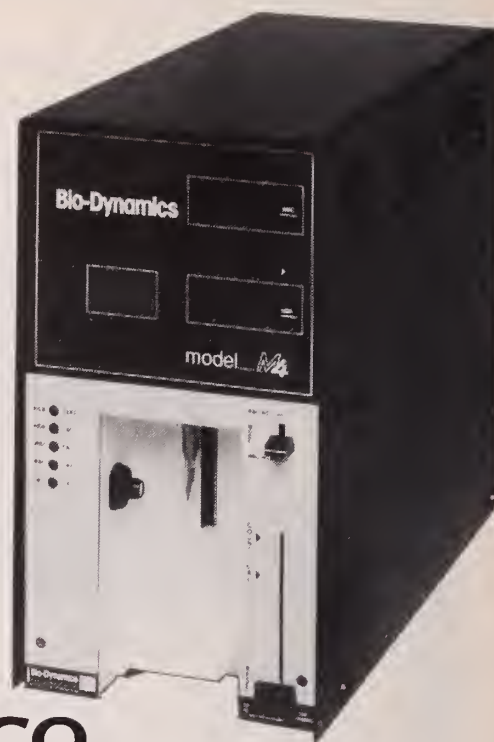
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Medical Malpractice — 1985

M. MARTIN HALLEY, M.D., J.D.,* *Topeka*

THE MALPRACTICE problem is again approaching crisis proportions, impacting upon patients, competent providers, law, the insurance industry, the economy, government, and society. Primary factors, frequently obscured, are patient injuries, a by-product of modern health care, sometimes resulting from providers' negligence. The analogy to industrial injuries is apparent. The tort system, based on "fault," continues to be ineffective for reasonable and prompt compensation. The same system was condemned as anti-social and oppressive during the development of workers' compensation programs.

Innovative concepts increasingly recognize the health care injury, and compensation and assistance in rehabilitation without the proof of fault. Model programs are available in the New Zealand Accident Compensation Act, The Swedish Patient Injury Insurance Plan, and Workers' Compensation in the United States. The ultimate solution is replacement of our fault and tort law structure with an innovative no fault compensation system.

The problem of medical malpractice, more accurately referred to as medical professional liability, remains unresolved. Health care delivery crises again appear imminent, signaled by increasing claim frequency, rising claim severity, and escalating insurance premiums. Basic issues involved in the continuing controversy are generally not well understood, or may be distorted by the intensity or self-interest of the parties, creating difficulty in objective evaluation. Adequate data are not easily available, so that analysis producing meaningful information for decision making is difficult.¹⁻⁵

What is the medical malpractice problem? Where does it impact? How can it be solved? These questions will be addressed in this discussion in terms of past and present developments in order to identify a potential long-term solution.

* Dr. Halley, a cardiovascular and thoracic surgeon, is a graduate of Harvard Medical School and Washburn University School of Law. He has contributed to medical and legal publications, and has participated in local, state, and national programs. He has served on various committees related to professional liability, and is on the Editorial Board of *The Journal of the Kansas Medical Society*.

Address reprint requests to Dr. Halley, 40 Medical Arts Bldg., Topeka KS 66604.

The Problem

In 1971, the pervasive nature of the malpractice problem focused national attention on the subject, not due merely to the rising volume of malpractice claims, but due to concern for their potential impact on the entire health care system. A presidential directive convened a commission on malpractice, and included the following observation: "The consequences of the malpractice problem are profound. It must be confronted soon, and it must be confronted effectively, but that will be no simple matter. For one thing, we need to know far more than we presently do about the complex problem. . . ."⁵

Today we do know more about the problem, but reasonable minds still differ on its precise definition. It is not primarily a problem of substandard health care practices, solvable through risk management or disciplinary action against providers. It is not primarily a controversy between physicians and trial attorneys, or between health care providers generally and the legal profession, although reports and news media coverage of events may convey this interpretation. It is not primarily a problem of the insurance industry, manifested by rising premiums, although this sector is certainly involved, as are the economy, state legislatures, state regulatory agencies, and the federal government. It is primarily, however, a problem of patient injuries, real or imagined, arising out of or in the course of health care delivery, and at times resulting from health care providers' negligence.⁵ It is a problem of personal injury to patients in the environment of high technology-modern health care, multiple treatment modalities and drugs, an astronomical number of decisions or individual judgments for delivery or non-delivery of care, and the occurrence of sub-optimal or bad results or treatment failures, sometimes in patients who formerly might not have survived.

The problem is ultimately one of society, wherein the analogy of industrial injuries to health care injuries is increasingly apparent. The former presented as a by-product of industrialization, and because of public concern for workers and their families, resulted in legislation for the protection and compensation of workers against the special hazards intrinsic in an industrial society, first in Europe, and subsequently in the United States.⁶ The latter are an increasing hazard to patients, as an unfortunate by-

product of modern health care. Societal concern should, therefore, result in a solution providing protection and compensation — reasonable and expeditious and without the uncertainties related to determination of fault — for patients against the special risks intrinsic in health care delivery.

Historical Perspective

The Code of Hammurabi in 2250 B.C. prescribed penalties for physicians who caused loss of life, or loss of an eye. Several English malpractice cases appeared in the 1700s, and the first United States case was reported in 1794. The incidence of claims was then relatively insignificant for nearly 140 years, but in the decade 1930-1940, the number of claims rose tenfold. Another tenfold increase occurred during the decade 1940-1950, and since 1950 the trend in claim frequency and severity has continued.⁷ In 1975, a national crisis in insurance availability and cost resulted in various legislative remedies involving tort law reform, frequently combined with disciplinary measures for health care providers. Liability insurance thereafter again became available. Claim frequency, severity, and insurance costs are presently escalating, and settlements, awards, or judgments in seven figures are increasingly common.

Paradoxically, the phenomenon of increasing malpractice claims and awards is occurring in a society where health care practice and achievement have attained heights previously unimaginable, and where great scientific and technological advances continue. The phenomenon is not limited to health care, but is one segment of personal injury litigation prevalent in the United States today, which includes automobile liability, product liability, air and rail liability, home owner liability, as well as professional liability generally. Medical malpractice is, therefore, a part of a general trend in tort litigation, although a number of more specific causes are as follows: diminished rapport with patients accompanying the technological and medical advances; unrealistic consumer expectations and consumer frustrations; an increasingly litigious society; an increasing number of highly skilled and increasingly specialized attorneys working in the context of the contingent fee; increasing emphasis in law school curricula upon medical malpractice; news media publicity for all kinds of medical affairs; and the influence of increasingly large awards, judgments, and settlements.

Pro-plaintiff changes in law in recent years have also been significant. These changes include abolishment of the doctrines of charitable and gov-

ernmental immunity for institutions; expansion of the locality rule for medical standards and the rules for expert witness qualification; findings of oral guarantees; long statutes of limitation; liberal application of the discovery rule; application or extension of the doctrines of *res ipsa loquitur* and informed consent; liberalization of doctrines relating to prenatal or perinatal injury; and extension of concepts of mental suffering or emotional disturbance. Finally, pro-plaintiff changes include court expansion of tort law doctrines into strict liability to compensate an injured plaintiff in the absence of demonstrable fault, where the defendant, through the use of insurance, is the more responsible person. The courts are hereby compensating individuals who suffer damages through no fault of their own by assessing damages against health care providers. Thus they "spread the risk," predicating compensation not upon the liability of an individual defendant, but upon the existence of a "deep pocket" or an insurance fund able to pay the compensation.^{8, 9}

Basis of Liability

Liability in the present tort system is frequently based on negligence although other legal theories may be applied. Negligence actions involve (1) a duty as arising out of the physician-patient relationship; (2) breach of this duty by deviation from the standard of care; (3) damage to the plaintiff; and (4) a causal relationship between the breach of duty and the damages. The legal concept of the standard is stated in court decisions as the duty of the physician to possess and exercise that degree of care and skill that is expected of a reasonably competent practitioner in the same class, acting in the same or similar circumstances.¹⁰

The standard of care may be visualized graphically as the density function of a continuous random variable, the bell-shaped curve, a probability distribution applicable to biologic variables such as cognition, decisions, and actions. Sixty-eight per cent of behavior will fall within one standard deviation of the mean, 95 per cent within two standard deviations, and 99.7 per cent within three standard deviations. Assuming the separation of relatively good and relatively bad practice to occur at the mean, a predictable percentage of actions or decisions will be good, better, bad, or worse. Thirty-four per cent will occur within one standard deviation, 13.7 per cent will occur within one and two standard deviations, and 2.3 per cent will occur within two and three standard deviations on either side of the mean. It follows that a practitioner, no matter how knowledgeable, how competent, or how skillful,

will make sub-standard decisions or perform sub-standard acts on a statistically predictable basis. The performance curve may additionally be shifted unfavorably by other circumstances such as relative cognitive ability, decisions or actions outside the practitioner's major expertise, fatigue, distraction, over-extension, behavior of assistants or others, as well as by technical, environmental, or patient factors. Even if all decisions or actions are acceptable, a number of bad results will be similarly predictable, and "fault" may be found through application of strict liability. The bell-shaped curve thus illustrates a major defect in the tort system, presenting competent practitioners with unfavorable probabilities or "negligence" and, on the other hand, requiring injured patients to prove the deviation from the mean and "fault." The combined effects of this and other defects of the tort system can be summarized as follows: there is no objective standard of liability; there is no definite measure of compensation; the entire process is susceptible to subjective considerations; the cost of litigation is high, in expenses and attorneys' fees; there is no restraint mechanism to litigation; there is no encouragement for prompt settlement; and finally, the system encourages and facilitates ever increasing awards.

The same traditional tort system applicable to industrial injuries prior to the enactment of workers' compensation statutes was condemned as anti-social and oppressive in 1909-1910 reports to the State of New York legislature. Investigating commissions unanimously concluded that (a) a large portion of all fatal and non-fatal injuries remained uncompensated, (b) the sums actually paid were frequently inadequate token compensation, (c) recoveries were obtained only after protracted litigation, (d) the attorneys of the injured workers retained a large share of the sum actually obtained, and (e) an undue portion of the premiums paid by industry went to insurance companies for profits and administrative costs and was thus socially wasted.⁶

Impact: Health Care Consumer

Patient injuries, real or imagined, are prime factors in the malpractice problem,⁵ which is additionally affected by other causes. A number of generally beneficial recommendations have been made intended to minimize such injuries, but none can be expected to significantly abate the problem. There is no evidence that more or better education in our already lengthy health care programs, expanded disciplinary procedures against health care providers, more or better post-graduate education, increased emphasis on hospital licensing, hospital

staff regulation, or other measures to encourage professional competence would have a significant impact.

In the patient's view when all is said and done, health care is a necessity and a right, but includes an inherent risk of injury. Compensation and rehabilitation must, therefore, be emphasized, but both are slow and uncertain under the present tort system. Injured patients, when compensation occurs, ultimately receive 20 per cent or less of the insurance dollar. The overall effect appears to be a wind-fall for a few patients, large rewards for a few attorneys, and income for insurance companies, defense attorneys, and others involved in the system.

Impact: Health Care Providers

The impact on health care is not only a matter of financial burden to providers, or increased cost to the public which ultimately pays both the direct costs of insurance and the indirect costs of defensive practices. The problem has been noted to touch every facet of our health care delivery system, including costs, patterns of medical practice, forms of treatment, the distribution of health manpower, the relationships between physicians and patients, and even confidence in equal justice before the law.⁵

Undesirable effects occur in the physician-patient relationship, since physicians must increasingly view each patient as a potential plaintiff. Widespread defensive practices incur additional inconvenience, cost, and risk to patients in the form of longer or earlier hospitalizations, an increased number of procedures or tests, recommendations against some procedures for legal rather than medical reasons, more consultations, early referrals to other physicians, stricter limitation of practice, withdrawal from emergency service, or early retirement of experienced physicians. Another significant problem may be physician dysfunction subsequent to the filing of a claim, or during trial. On the other hand, beneficial effects of defensive practices have been noted, since these may also be good patient care, and quality control benefits for health care through the threat of tort litigation have been suggested.

Yet another area of impact is the increasing participation by physicians or other health care providers in claim review for attorneys or as medical witnesses. A recent estimate based on analysis of national advertising material indicates that 3000 physicians are active in this process. Remuneration is substantial: \$500 for chart review, analysis or report; \$600 for depositions; and \$3000-\$5000 daily, plus expenses for court appearances. Thus the alleged medical "conspiracy of silence" — once a

chronic complaint of the legal profession concerning difficulty in obtaining medical witnesses — has been replaced by vigorous marketplace activity, with numerous physicians competing for the opportunity to participate in a lucrative field. Availability of abundant medical testimony and assistance in case preparation is believed to be one of the catalysts that has opened the floodgate of professional liability litigation in the medical field.¹¹

Impact: The Legal System

The legal system, consisting of attorneys, courts and law, has been a fundamental factor in the present malpractice problem, most importantly through the expansive application of the fault and liability concepts of tort law. Attorneys and judiciary will continue to be important in the developing efforts to restructure the tort system, and can be expected to resist change in terms of fault, liability, adversary and litigation, concepts deeply ingrained in Anglo-American law. Law students and attorneys are firmly attached to the adversarial process which requires parties to “battle” to reach truth and justice, a process that essentially renders only a victor. Law students and attorneys are less familiar with the terms negotiation, settlements, mediation, and compensation without fault. Therefore, the “fault” frame of reference continues to be the legal profession’s response to many societal problems, as contrasted to the “compensation” system based on a foundation other than “fault.”¹²

Certain other factors involving the legal system deserve mention. The increasing number of attorneys — which has resulted in manpower for specialization and increasing expertise — has been suggested as a major factor in the escalation of the malpractice problem. Attorneys — both plaintiffs and defense — with special interest in this field, contribute not only time and skill to legal issues, but exercise considerable legislative influence to actively or passively oppose changes in a framework that has identified health care with other major target defendants. The legal system thus professes to protect the rights of patients and strives for injury compensation within the present tort law, but these objectives are at times obscured by seemingly inappropriate tort law results, as well as by highly visible controversies involving opposition to legislative reforms of tort law structure.

Impact: Insurance

The insurance industry at the time of the 1975 crisis was in a state of near collapse, manifested by carrier withdrawal from the marketplace and by in-

creasing premiums. State legislatures then enacted a variety of tort law modifications, and frequency of claims declined for three years, but claim severity continued to increase. In the 17 states where a financial limitation was placed on awards, and in the 16 states where the collateral source rule was repealed or modified, claim severity was reduced, but no effect on frequency was noted. No other substantive legal reform was found to have statistical significance in reducing either the frequency or the severity of claims.¹³

In Kansas, the Health Care Stabilization Fund, established in 1976 under the Health Care Providers’ Insurance Availability Act, was intended to be accompanied by a limitation on total awards.^{14, 15} The limitation, however, was never seriously considered by the legislature, and by 1983 the fund was actuarially insolvent. Legislative intervention was required: basic insurance coverage requirement was increased, excess coverage available from the fund was limited, and the levy of the fund surcharge was changed to an accrual basis. As a result, health care providers’ insurance premiums increased substantially.

The Kansas Insurance Department reported on June 30, 1984, that awards against the fund totaled \$22,222,605. Ninety-five awards had been paid with an average payment of \$233,912. A total of 710 cases had been filed, and 365 remained active. Most significantly, there had been eight claim awards, judgments, or settlements in the million-dollar range in the past 15 months. The increasing exposure of the fund, together with a continuing unfunded liability, may necessitate additional corrective legislation, as well as studies of insurance alternatives to its continuation.

Impact: Economy

The economic impact of malpractice is difficult to measure, although it is generally believed to be substantial. Present emphasis on health care cost-control focuses attention on this aspect of an industry accounting for 10.9 per cent of the gross national product. A 1983 report¹¹ estimated direct annual malpractice costs — the insurance premiums paid by physicians — as \$1.75 billion, but could not estimate hospital premium costs, which included liability premiums, risk management programs, and other miscellaneous items. Indirect costs, attributed to defensive medical practices, were estimated as \$15.1 billion, but a number of these practices may not be strictly defensive. Current direct costs are reported as \$3.5 billion, and indirect cost estimates range up to 30 per cent of total health care expenditures.

Impact: Government

State legislatures have been, and continue to be, the arena where reforms must be obtained, but remedies have been variable and have not resulted in a long-term solution. In Kansas and other states, legislation has produced only "piecemeal" reforms¹⁶ that have principally resulted in continuing availability of liability insurance.

The current legislative program of the Kansas Medical Society again consists of tort reform proposals. It will include proposals for limitation of total awards, limitation of pain and suffering, collateral source revision, modification of attorney's fees, itemization requirements for verdicts, reduction of the judgment interest rate from the current 15 per cent, allowing periodic payments to expire at the death of a plaintiff, and a procedure providing incentive to early settlement. Legislative consideration of these proposals will require concerted action by health care providers, including coalitions with other interests affected by present tort liability problems. Other requirements are general agreement on the program and its objectives, adequate funding, news media exposure, public education, legislative information programs, individual contacts with legislators, and perhaps a governor's task force to address important issues.

At the federal level, interest in medical malpractice is inevitable, since the government is the largest single purchaser of health care services. In the past, this interest has been passive, but in April 1984, an "Alternative Medical Liability Act" was introduced in the House by Representatives Moore and Gephardt,¹⁷ incorporating previously published recommendations,¹⁶ and was referred to the Committee on Ways and Means. This bill provided for settlement of malpractice claims arising in programs established under federal law. The central feature permitted a health care provider potentially liable for personal injury to tender compensation for the claimant's net economic loss, and by this act, to foreclose tort law litigation.

Medical opposition to the bill focused on its doubtful effect on defensive medical practices, the possible cost impact of an increased number of claims, problems of court involvement, and issues related to provider decisions, third party joinders, and federal intervention. The appearance of these concepts at federal level may signal a more active posture, and may serve to encourage state solutions in the hope of avoiding further federal action.

Discussion

Efforts to resolve the malpractice problem in the

United States have generally involved insurance alternatives or tort law modification, as well as recommendations for preventive action in the health care industry. Insurance programs are of obvious importance, since availability and reasonable premium structure are essential to continuing health care practice. Tort law modification has been difficult to obtain, and has not proven significantly effective, since available data indicate that only total award limitation and collateral source modification have produced measurable short term mitigation. Preventive programs are important, but an irreducible number of injuries will nevertheless occur.

Innovative concepts are, therefore, assuming increasing significance. Their overall thrust is the evolving recognition of the health care injury arising out of modern health care delivery, and the increasing consensus that compensation for such injury, and assistance in rehabilitation, should not depend upon the proof of fault.¹⁸⁻²¹ Definition of the injury, or compensable event, continues to be the major challenge in several available studies.^{22, 23} Initial concerns about unfavorable cost impact are being re-evaluated in the light of present monetary values and enormous awards. Other components of a compensation system include the measurement of damages, the form or amount of compensation, the source of compensation funds, case-disposition mechanisms, fund collection and disbursement, and methods of dispute resolution. Three apparently successful injury compensation systems, the New Zealand Accident Compensation Act,²⁴⁻²⁷ the Swedish Patient Injury Insurance Plan,²⁸⁻³⁰ and the Workers' Compensation System in the United States,^{6, 31} are available models for analysis and comparison.

New Zealand, since 1974, has defined personal injury by accident to include "medical, surgical, dental, or first aid misadventure." The program merges workers' compensation and automobile protection, and adds coverage for victims of other accidents. Common law actions for negligence are precluded to the extent that an injury is compensable under the no fault system, but it is not yet clear which cases will be compensated and which will be litigated. Financing is by levies on employers and self-employed persons, levies on owners and drivers of motor vehicles, and money appropriated by parliament.

Benefits include medical care, transportation to the physician or hospital, funeral expenses, awards for permanent loss or impairment of function, payment for lost earnings, and limited awards for disfigurement or pain and suffering. In exchange for the new program, the injured person has traded the du-

bious advantage of litigation of torts for a quick and informal administrative procedure. He has traded the possibility of a large award for a more certain, modest payment for injuries, limited pain and suffering, and assurances of income maintenance.

After four years of operation, this innovative program appeared to be working well. Claims were processed rapidly and routinely, and with an acceptable administrative cost. Few claims were appealed beyond the initial level and most were paid without attorney involvement. One of the major difficulties was ascertaining the range of coverage intended by the statutory words.

Sweden, in 1975, established a patient insurance program after general realization that few medical malpractice claims resulted in compensation for the patient. The primary goal was to create a provider-financed scheme for compensating victims of significant medical injuries in three categories: (1) an injury that occurred as a direct consequence of examination, medication, and treatment, and that was not associated with known risks, but excluding injuries or illnesses likely to have arisen irrespective of care rendered; (2) an injury that occurred as a result of incorrect diagnosis or an incorrect interpretation of symptoms, that is, procedures not reflecting generally accepted medical practice; and (3) an injury that occurred as a result of a sudden external event within the health care institution or during transportation by ambulance.

Negligence was discarded as a standard of payment, and the mechanism for discipline of providers was separated from the compensation program. Insurance coverage to pay claimants is purchased from a pool of private carriers by private and government-employed health care providers. Claims are submitted to an insurance office, and most are then paid directly. Appeals are possible to a claims panel, and thereafter may be submitted to arbitration. The patient initially retains the right to proceed in the court system, or may file a complaint with the National Board of Health and Welfare.

Objectives of the program were to overcome some of the disadvantages in the prior fault based system, and to accomplish speedy resolution of claims without the adversary process and litigation. Results of the program do not indicate generation of unduly large numbers of claims, and do not indicate extensive investigation of claims or contests in the level of awards. Review of the program's development will establish the extent of these results, as well as the effects of separating the compensatory side of medical malpractice from the disciplinary aspects.

Workers' Compensation, the oldest branch of

modern social insurance, became part of the European legal system long before its acceptance in the United States. Beginning in Germany with the enactment of an Employer's Liability Law in 1871, a number of other continental powers adopted industrial accident insurance acts before the turn of the century. England followed with the Employer's Liability Act of 1880. In the United States, in 1909, Montana enacted a state compensation system for the coal mining industry. Subsequently in 1911, the largest number of state statutes were enacted, and in 1971 constitutional barriers were removed when the Federal Supreme Court upheld the three existing types of compensation laws. The adoption of a compensation act by Mississippi in 1948 made the system universal.

Thus, in a span of approximately 80 years, the inadequacies of the tort system were gradually corrected so that the victims of industrial accidents and their families were adequately and promptly protected. New legal principles were needed, but legislators were slow to grasp this necessity. These new principles established that the great bulk of work accidents should be regarded as part of the unavoidable loss of modern industrial operations, and should not be approached with concepts of fault. The accident toll presently in American industry is nearly 43 million working days annually; at least 16,500 deaths occur each year through routine industrial operations; and accidental limitation of earning capacity involves more than 2 million other workers. Compensation is payable according to a definite scheme. Payment is secured by employers through private insurance, state funds, or self-insurance. Fault has been eliminated. The compensation represents a compromise in which each party surrenders certain advantages in order to gain others more important to him/her and to society. Employers give up the immunity they would enjoy in cases where they are not at fault, and employees accept a smaller, but certain and prompt compensation.

This system appears to have resolved the problems of industrial accident compensation, and no serious argument has appeared for return to the tort system. Should it not be asked why the same scheme is not equally appropriate for many other injuries presently administered under the traditional fault system of tort law?³¹

Conclusions

Tort law approaches to the medical malpractice problem have not resulted in a permanent solution due to the inherent disadvantages of the fault approach. In health care, profoundly negative

effects involve both delivery and cost. In law, plaintiff changes have further extended tort law application into the realm of strict liability, and segments of the legal system have effectively opposed most legislative reforms. In government, legislation to date has provided only short-term relief, has been accompanied by disciplinary measures for providers, and has principally resulted in availability of insurance to pay the ever increasing awards and settlements.

For the patient, high quality health care has been accepted as an individual right in modern society, accompanied by the growing awareness that such care includes inherent risks of personal injury related to the health care process, rather than to the underlying illness. For society, the concept of the health care injury, strikingly similar to the industrial accident, appears increasingly attractive as a long-term solution of this complex problem, awarding compensation based on no fault principles.

Many parallels exist in the evolution of the malpractice problem and in the development of workers' compensation systems, as well as in the development of present no fault compensation programs for health care injuries in New Zealand and in Sweden. The ultimate solution is thus on the horizon, as the replacement of our venerable fault and tort law structure by an innovative no fault compensation system.

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WAYNE T. STRATTON, J.D.,* *Topeka*

LAWSUITS that arise from the physician patient relationship are as old as American legal history. As early as 1794, a physician was held liable for improperly performing a mastectomy.¹ Legal principles governing medical malpractice actions in Kansas were established by the Kansas Supreme Court in 1870.² However firmly entrenched in legal history, the rights of a patient to sue for medical malpractice and the remedy the patient will be granted have been questioned. This inquiry stems from a mid-1970s medical malpractice crisis and continuing increases in the amount of litigation and the size of jury verdicts.

More medical malpractice cases have been filed in the past 20 years than in the entire preceding century.³ In the late 60s and early 70s medical malpractice claims rose by 81 per cent, and the average size of such claims doubled.⁴ Thirty years ago such suits were rare because the practice of medicine was simpler and because the physician was viewed as holding a higher position of trust and respect by society as a whole. A poor outcome was more likely to be attributed to an "act of God," rather than physician negligence. Today, physicians must meet a higher standard. Patients have developed greater expectations of beneficial outcomes along with a greater awareness of their ability to pursue a legal remedy. Medical practice has become increasingly complex resulting in a decline of patient trust and provider-patient rapport.⁵

Although litigation has increased for all types of claims, those against health care practitioners and manufacturers have outpaced all other types of personal injury lawsuits. In 1969, nationwide, one insured physician out of every 23 was the subject of a filed claim. In Kansas, one physician out of every 33 was faced with a claim. By 1974, these figures had risen to one out of ten insured physicians nationwide, and one out of 18 in Kansas.⁶

The increased frequency, severity, and unpredictability of claims against health care providers precipitated an insurance crisis in the mid-70s. In 1974-75, many physicians found their malpractice premiums increased by as much as 300 per cent. Insurance companies restricted coverage and many com-

panies withdrew from the market. Providers reacted by passing on costs of the increased insurance to their patients, by attempting to avoid claims by practicing defensive medicine, by avoiding practice in high risk specialties, by moving to states where insurance was more affordable, and by giving up medical practice altogether. Legislatures responded to the possibility that affordable health care would no longer be available in their states by enacting new laws.

Generally, the mid-70s reforms were designed to restrain the scope of the health care provider's liability, reduce the size of awards, and discourage frivolous lawsuits so that insurance companies could accurately predict awards and regulate premiums accordingly. Primarily, the legislative changes included the imposition of limits on the amount a patient could recover for any act of medical negligence, limited the provider's liability to a fixed amount, reduced the statute of limitations to eliminate the "long tail" of liability, abrogated the collateral source rule, authorized periodic payment of large awards, established medical screening panels, encouraged arbitration, and set up patient compensation funds. Of the 1975 tort reforms, caps on awards and mandatory offset from collateral sources appear to have had the greatest impact on the severity of claims.⁷ Malpractice insurance did become available after the reforms were enacted, but on the whole the legislative changes have not really proved effective in reducing either the frequency or severity of claims.⁸ A 1980 study of closed claims by the National Association of Insurance Commissioners concluded that the reforms did not resolve the malpractice crisis. This conclusion has been supported by current statistics. The cost of defending and bringing suit for medical malpractice has increased by 73 per cent since 1976. Between 1978 and 1983, the number of claims filed has increased 5 per cent. From 1975 to 1983, the mid-point of jury verdicts awarding damages against physicians and hospitals doubled, and for physicians alone, the figure tripled.⁹

The number of million-dollar verdicts rendered in medical malpractice lawsuits and product liability cases substantially outpaced the number of million-dollar verdicts rendered in all other personal injury actions. Forty-three per cent of all million-dollar verdicts are in these areas. In medical malpractice

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cases, the greatest number of million-dollar verdicts are awarded in wrongful death litigation and litigation centering around birth-related incidents.¹⁰

Not only are higher awards being rendered, but more claims are being filed. According to the American Medical Society, "no segment of litigation has grown as rapidly in the last 15 years as claims arising from health care."¹¹ Three times as many providers were sued for malpractice between 1978 and 1983 as had been sued prior to that time. Court statistics for Kansas show a short-lived decline in the number of medical malpractice suits filed after enactment of the reforms, then a trend toward increased filings. During the same period, automobile injury cases showed a similar decline and rise, but they did not rise above the previous number of filings as did medical malpractice cases.¹²

From January 1 to May 19, 1984, more than 145 claims were made against the Kansas Health Care Stabilization Fund (HCSF). This is more than the annual number of medical malpractice cases filed between 1977-1979. Nationally, the number of malpractice claims filed and the increase in awards differs from all other personal injury litigation, except product liability actions, where the number of cases filed and the size of awards has remained relatively constant from 1973 to 1982.

As a result of the increased frequency and severity of medical malpractice litigation, insurance rates are on the rise. In Kansas, rates for medical malpractice insurance doubled last year.¹³ The Kansas increase was precipitated by the potential inability of the HCSF to pay all claims pending against it, the necessity of raising the basic insurance coverage limits, and the implementation of premium increases by the insurers. Moreover, many providers are now required to purchase excess coverage because a \$3 million limitation on recovery from the HCSF was enacted by the 1984 Kansas legislature without a corresponding limitation on provider liability.

The Kansas insurance increases are representative of a national trend, precipitating a new medical malpractice crisis.¹⁴ In the 70s, the problem was one of insurance availability; in the 80s, the problem is the cost of insurance and the expense of personal liability.

The origins of the pending crisis vary according to the commentator's role and interests. As was claimed about the mid-70s crisis, some commentators contend that the present situation is the result of insurance company manipulation — that no "real" crisis exists. Persons advocating this position contend that malpractice insurance premiums account for only a fraction of a health care provider's annual

gross income.¹⁵ Nonetheless, it is apparent that in high risk fields, premiums run as high as \$50,000 annually. Of the amount paid for premiums, the majority of money is spent for claims resolution rather than actual compensation to injured plaintiffs.¹⁵

Health care providers echo the complaints of a decade ago contending that despite legislative changes, the frequency and severity of claims has made insurance unaffordable, has forced physicians to abandon high risk specialties, and has forced the practice of defensive medicine. The result is that more providers will retire early; fewer providers will perform high-risk procedures or specialize in high-risk areas; care costs will increase; and the availability of care will decrease. Legislative reforms to insure fair compensation to injured plaintiffs in appropriate circumstances and to insure the financial security of health care professionals are necessary to cure the underlying causes of the malpractice crisis.

The increase in medical malpractice litigation is obviously due to many factors. It is interesting to compare the impact of this litigation with automobile accident and product liability lawsuits. Almost all jurors drive and can readily identify with drivers in automobile accidents. Almost every consumer has at one time or another purchased an unsatisfactory product. However, jurors do not practice medicine and tend to look upon medical matters as a mysterious science. When the results of medical treatment are poor, the jury relates only to an ill or infirm plaintiff, with only a limited understanding of the defendant health care provider's actions. A Rand Institute study affirms this proposition. The study found that the legal standard of negligence often has little to do with the outcome of a malpractice case. The degree of injury, its severity, and economic impact determine recovery.⁶

Mandatory automobile insurance has increased public awareness of undeserved verdicts in cases such as those described above. No similar constraint operates in medical malpractice. Juries tend not to equate high health care costs with high malpractice insurance premiums which inevitably result from undeserved verdicts. Improved claim handling and damage thresholds imposed by no fault automobile insurance statutes have been factors in curtailing increased automobile litigation. Similar statutes do not operate to curtail medical malpractice claims. Additionally, the skill and knowledge of the plaintiff's bar has continued to increase, and the most skillful attorneys are drawn to fields of practice offering the highest rewards.

It is submitted that medical negligence has not

increased during the last 20 years. The change has been in the perception and values of society as a whole coupled with the performance of more intricate and potentially dangerous medical procedures. To successfully effectuate any change toward required reforms, mechanisms must be introduced that change the perceptions of society by increasing its awareness of all aspects of the subject of medical malpractice.

The argument has been raised that the threat of litigation forces health care providers to be cautious in choosing treatment alternatives, and that the present tort system therefore serves as a continual deterrent to medical negligence. However, some legislative enactments in the mid-70s did relate to better surveillance of the medical community in an effort to insure quality health care. Peer review committees, continuing education requirements, and long-range study programs were among the best reforms of that era.¹⁶ Kansas has provided for the establishment of peer review committees and for confidentiality of their records. A disciplinary system for review of complaints filed against providers has been established and tied into provider licensing regulations.¹⁷

If, as in Kansas, the present tort system is not the only way to deter acts of medical negligence and to compensate injured patients, then tort reform is crucial to reduce the frequency and severity of claims. Real tort reform begins with a reasonable definition of medical negligence and extends through case presentation. Suit advantages for the plaintiff and the defendant which became unbalanced in the late 1960s need to be put back in perspective, along with jury sympathy.¹⁸

One area where reform would be effective revolves around the legal standard of care applicable to health care providers. Since adverse consequences can result from proper treatment, often what is questioned is a professional judgment based upon the provider's perception of what would be the most beneficial course of treatment for that particular patient at the time the patient sought treatment. The legal definition relating to standard of care should accurately reflect the context in which that judgment was given.

In an ordinary action to recover damages for personal injuries, it is sufficient for a plaintiff to show what the defendant did and the circumstances surrounding the defendant's actions. If the defendant acted reasonably, then the plaintiff fails to recover. However, a lay jury has no expertise to determine the context of a medical malpractice defendant's actions, and expert testimony must be used. Under current standards, almost any expert can testify re-

garding any situation. Expert testimony is now "big business," and plaintiffs have little trouble finding an expert to testify that the defendant health care provider's conduct fell below the standard practice.¹⁹

Some states have adopted provisions that would insure that more of the total premium dollar is used for compensation by limiting the fee a plaintiff's attorney can recover in a medical malpractice lawsuit. The court either limits contingency fees recoverable per a statutory scale, or reviews the fee to be certain it is reasonable. Contingent fees are a proper and appropriate method of compensation in certain cases. In medical malpractice lawsuits, such legislation discourages frivolous claims and enables plaintiffs to receive the majority of their awards.

Liability caps have been enacted as a means to restore balance to the tort system. In states that have held caps constitutionally valid, they have proven effective in reducing the effects of the malpractice crisis. Another effective means of reducing the severity of claims is to tie the patient's recovery to his actual losses. Limiting non-pecuniary damages and allowing compensation from other sources to be deducted from the award prevents over-compensation because of jury sympathy and prevents double recovery.

In addition to the somewhat traditional approaches to tort reform, new compensation systems are being investigated to mitigate the potential effects of the malpractice crisis. A no fault insurance compensation system similar to automobile no fault systems has been proposed. A workers' compensation type of system might be possible. However, such systems pose practical problems primarily in defining a compensable event. The 98th Congress has held hearings on a proposed system that offers plaintiffs a trade — prompt payment of compensation in exchange for the right to sue for pain and suffering and punitive damages.²⁰

The issues surrounding medical malpractice litigation and the continued delivery of health care services are complicated. The topics raised are complex because of the multiplicity of issues, the high level of competing interests, and the emotional atmosphere surrounding such litigation. Reform will not be simple. Ultimately, the legislature must resolve the new crisis by providing an equitable compensation system while insuring that health care remains available and affordable.

Acknowledgement

Marta Fisher Linenberger, a third year law student
(Continued on page 354)

SB-646 Revisited

HOMER H. COWAN, JR., *Fort Scott*

THE MEDICAL malpractice crisis, born on the west coast, had worked its way inward to the heart of the country — Kansas. It was 1975, the year of black armbands worn by Kansas physicians and hospital administrators to draw attention to their plight — the high cost and restricted availability of insurance. Some physicians left the state. It was a crisis.

SB-646 was an attempt to address the problem in the Kansas legislature, a legislative proposal that, coupled with a number of other changes, was designed to tone down the tort exposure. This was a bold legislative package worked out between some segments of the legal profession, the medical professions, and the insurance industry under the guidance of Insurance Commissioner Fletcher Bell. Senator Wes Sowers guided the proposals through the legislative process, and in 1976, SB-646 became law (*Figure 1*).

This law established a residual mechanism called "The Plan" that wrote all risks not accepted by the private market. The basic coverage of 100/300 was supplemented by "The Fund," financed by surcharge on premium, which provided *unlimited* excess coverage. The law provided that a surcharge could not be applied when The Fund accumulated \$10 million.

But SB-646 was defective from the start. First, The Fund (the re-insurance layer) offered *unlimited* liability. Basic principles of insurance require limits of liability in order to price the product. In 1976, a request to the marketplace to price unlimited liability would have produced no quotes — or quotes that would have been unacceptable.

Another defect was the base. For insurance to be affordable, the base must be broad enough to support the claims of the few. There has always been and still is a question of the medical base in Kansas being able to support relatively few claims if any of them resulted in the million-dollar jury verdicts that were becoming commonplace in other states. In 1976 Kansas had not experienced any judgment approaching such a figure, and there were those who said a Kansas verdict would never be that high.

During the first few years after 646 became law, loss ratios were embarrassingly low, raising the question as to whether the insurance industry had

been using excessive rating bases. Experience was so good that the surcharge used to build the financial integrity of The Fund was dropped. Kansas physicians were, in effect, receiving unlimited coverage for the price of a 100/300 policy.

In retrospect, the reasons for the early success of the Kansas Health Care Providers Insurance Availability Plan are clear:

- The mechanism as set forth in the law was, by far, the most effective and simplest mechanism devised by any state legislature;
- The acceptance of "claims made" coverage allowed the mechanism to start with a clean slate; it did not buy into the front-end tail; and
- While the crisis seemed to subside, it was only temporarily deterred. The public was aware of the crisis, and attorneys felt juries might not be as generous as they had in the past; therefore, not many cases were filed in 1976 or 1977.

By 1982, cracks were appearing in the protective walls of SB-646. The mechanism was still working, but claims were now *maturing* under the claims-made concept. Maturity and inflation sharply increased claim and defense costs, and Kansas juries had forgotten about the crisis and were ready for the million-dollar verdicts long predicted by the industry.

And The Fund, the upper layer of unlimited liability, was now broke because it had not reserved for the maturity of claims. Had this Fund been an admitted carrier, it would have been shut down long before the coming of the second crisis!

In 1984, much to the credit of the Kansas medical

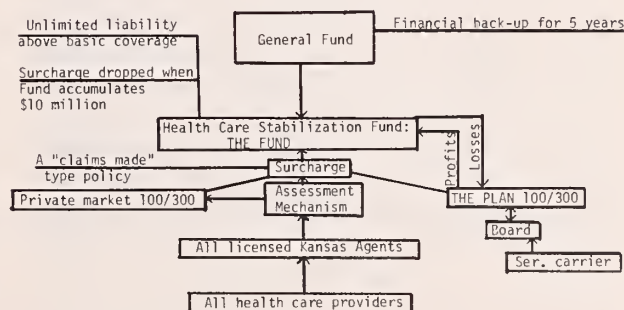


Figure 1. SB-646 enacted in 1976 (Article 3A-40-KSA-3401, The Health Care Provider Insurance Act).

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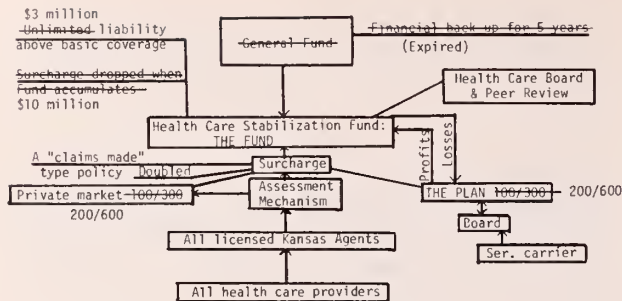


Figure 2. SB-646 as amended to become SB-507. 40-KSA-3412, paragraph D, exempts The Fund from payment of punitive damages. This language, contained in the original text of SB-649, was not changed by SB-507.

profession, physicians and hospitals bit the bullet, and with the guidance of the Kansas Insurance Department, SB-507 became law, amending SB-646 as shown in Figure 2. The amended act accomplished the following:

- Capped The Fund at \$3 million *per claim*, \$6 million aggregate;
- Removed the limit affecting surcharge;
- Doubled the surcharge;
- Doubled the basic coverage; and
- Created a Health Care Board and Peer Review, with confidentiality protection.

Unfortunately, like its predecessor SB-646, SB-507 is defective. The \$3 million per claim payable by The Fund is too large to be supported by the Kansas medical base. Those who need to purchase coverage in excess of \$3 million will find a lack of availability and high rates. Several million-dollar verdicts will once again jeopardize The Fund, even with substantial surcharges.

The remedy will not be popular, but to make the concept work, The Fund needs a cap of \$500,000 per claim. There are many physicians whose practice does not generate the explosive residual exposures. At most, low profile physicians would likely purchase no more than an additional layer of \$500,000 excess coverage. Others, whose practices are volatile, would likely purchase higher excess limits. But at this level, the entire excess market is much more competitive because the excess base would be broad enough to offer the coverage at a much lower rate. Excess coverage above \$3 million will not create the

needed base at the excess level.

In spite of some relief from tort liability exposure passed in 1976, the deterioration of the tort system continues at a record pace. The public is being educated by headlines, and the simple message is "For every injury there must be a wrong!" And courts continue to rationalize themselves to that conclusion. Any suit is permissible, and if there is a sympathy factor, "don't worry about the facts, ma'am."

In spite of massive advertising programs underwritten by the insurance industry to convey the message "Jury awards are paid by *you*," jurors still think in terms of insurance money being different somehow from their own. Therefore, a verdict of \$1-3 million is not only becoming common, but the public still thinks it's free.

During the months of October and November 1984, a \$15 million verdict and a \$5 million verdict were returned in a 30-day time frame. This is indicative of the social and legal climate that can be expected for several years to come.

How many \$3 million verdicts can Kansas physicians and hospitals fund by way of surcharge? How much premium can physicians pass on to the patient?

Punitive damages are in vogue; if the residual injury is not dramatic enough to command a large award, juries make it up with punitive damages.

Why are juries so unsympathetic to the medical and insurance professionals? Because the professionals are the ones who always ask to be excused from jury duty, often leaving people who are totally unfamiliar with business and the "pass through" mechanism that forms a part of the economy itself. How much more protection can the physician afford? How much more insurance must s/he buy, which in turn will be paid for by patients whose lives have been saved?

SB-507 is a good step, but more is needed. This bill will probably require dramatic amendments in the next few years unless there is a change in the social and legal climate of America.

One thing is certain: The Kansas medical profession is a courageous group. They will keep trying until they get it right. The entire insurance industry stands in awe of the medical integrity now on display, the willingness to shoulder a large monetary burden for the benefit of Kansas patients administered to by Kansas physicians and Kansas hospitals. Good luck!

The Expert Medical Witness

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PATIENTS AND physicians alike called it the “malpractice crisis” when it began in the mid-1970s. It was not a crisis of increasing malpractice, but rather that insurance to cover such possibilities was suddenly not readily available. A few legislative band-aids were applied, and physicians relaxed and went back to work. Now, in the mid-1980s, professional liability is again a full-blown crisis. This time it is cost that drives the crisis — cost in time as well as money to a health care system already overloaded by the costs of advancing technology. A shocking point could be made with statistics that show what every physician already knows — the risk of becoming involved in a malpractice claim or lawsuit, the sheer numbers of claims filed each year, the average settlement per claim, the average jury award per trial. However, a positive search for solutions is preferable to a negative approach.

One solution lies within the four elements that must be present for a medical malpractice lawsuit to be valid:

1. A standard of care must exist for the particular medical procedure or practice about which the allegation is made;
2. Expert medical testimony must establish whether a deviation from that standard of care occurred and whether such a deviation constitutes *negligence*;
3. There must be an identifiable injury; and
4. Expert medical testimony must provide the causal link between the specific deviation and the identifiable injury.

A standard, a negligent deviation from the standard, injury, and causation — all must be present for a lawsuit to go to a jury. Since legal action cannot proceed without cooperation from the medical community, it is fair to state that one factor propelling the professional liability crisis lies in the pivotal role of the expert medical witness. Therefore, it is crucial

for physicians not only to understand how the adversary system uses the medical expert, but also to know what responsibilities are involved in being an expert witness. A more equitable approach to the presentation of expert testimony can be suggested once these issues are clearly understood.

The Adversary System

When a patient consults an attorney with a claim of injury as the result of medical negligence, the attorney tends to base acceptance or rejection of the case on purely economic considerations. Plaintiff's attorneys are usually paid a contingency fee, *i.e.*, they do not get paid if they do not win because their fees are percentages (30-50 per cent) of the settlements or judgments. The argument is that out of pure self-interest plaintiffs' attorneys do not pursue cases that do not have a reasonable chance of success. To determine whether a case is meritorious, attorneys often employ medical analysts, who may be physicians, to make that judgment. Much of the medical expert's work is accomplished in this pretrial analysis of the merits of the case.

Once a case is filed with the court, it enters the discovery stage. *Discovery* is a term that encompasses all the pretrial legal procedures that have been instituted to ease the ever-increasing burden on the court's time. These devices eliminate surprises at trial that can lead to mistrials, allow all parties access to supporting material and witnesses, and encourage the parties to settle those cases of apparent liability. As part of this process, both plaintiff and defense attorneys can question those witnesses that the opposition intends to use to prove its case. Factual witnesses may not express opinions; they can testify only about facts and circumstances to which they were a direct party. Expert witnesses, on the other hand, are qualified by their education, training, skill, and experience to render opinions based on evidence from a variety of sources, such as documents and the testimony of factual witnesses.

The importance of the medical expert's testimony cannot be overemphasized. Without it the plaintiff has no case. As a representative of the scientific medical community, the expert witness presents his/her definition of the standard of care to the judge and jury. It is this testimony that establishes whether the defendant-physician deviated from that standard.

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And it is this testimony that constructs the causal link between deviation and injury.

In seeking out medical experts to analyze and support their clients' contentions, lawyers are advised that juries find local specialists to be more credible than those from out of town. However, plaintiff's attorneys have difficulty finding local experts who are willing to testify. Lawyers as well as physicians understand the self-destructive nature of testifying against colleagues who are seen every day, against those who might be a source of referrals, or against hospitals where one has staff privileges. Consequently, brokering out-of-town expert medical witnesses for plaintiffs' attorneys is big business. In fact, the sad truth of the matter is "that any plaintiff's attorney worth his salt can find a qualified medical expert who will opine, under oath, that the standard of care was something other than what the defendant followed."¹ Here are some sample advertisements from a recent issue of a magazine for trial lawyers:²

"We help attorneys win medical malpractice cases. . . . There is no charge or obligation until we have a supportive report from a qualifiable expert."

"Board certified medical and surgical specialists in all fields of medicine. Available for evaluation, conferring and testifying on medical malpractice litigation. Will travel."

"The right expert witness can make all the difference in your next medical malpractice case. . . . Specialized areas include medical and dental malpractice, hospital negligence, personal injury, workers' compensation, product liability and equipment failure cases. An evaluation for merit service is also available."

For medical professionals as well as the lay public in general, the hardest thing to understand about the law is the nature of the adversary system, to understand how a lawyer, like a good debater, can argue both sides of a dispute with equal skill and zeal. "The key is to realize that the law functions to make the gray areas of human interaction black and white, to determine who has presented the strongest, most believable case and not to pursue absolute truth."³ To work within this system, the expert medical witness must maintain impartial, scientific objectivity and not get caught up in the partisanship that is the heart and soul of the law.

Responsibilities

The greatest responsibility of the physician who agrees to give testimony as an expert is to convey the

message that a medically approved standard may encompass a number of different modes of treatment. The standard is neither static nor absolute. It is best pictured as a bell shaped curve, with ideal treatment at one narrow extreme and unsatisfactory treatment at the other narrow extreme.

Other responsibilities can be summarized, without going into great detail, in a list of dos and don'ts for the expert medical witness:⁴

Do

- Learn about the adversary system so you do not become a legal pawn.
- Spend an appropriate amount of time to thoroughly evaluate all medical records before you express an opinion.
- Demand to see all medical records, not just those the attorney deems appropriate.
- Acknowledge when assumptions are based on incomplete data.

Don't

- Confine your opinions totally to that of plaintiff or defendant.
- Render opinions in areas outside your specialty.
- Base your opinion on plaintiffs' recollections or attorneys' statements.
- Render absolute judgments based on assumptions derived from incomplete data.
- Assume that incomplete or poorly documented records are evidence that medical negligence or injury occurred during that particular time.
- Assume that injury resulted from medical deviation unless all other possible etiologies for the injury have been ruled out.
- Be afraid to change your opinion if new evidence comes to light.
- Become a partisan advocate.

Solution

The crux of the problem is that the expert medical witness has become an instrument of the adversary system rather than an impartial representative of the medical community. The plaintiff can find a board certified expert who will look at the record and the circumstances and swear that there was deviation from his/her definition of the standard of care that caused the injury. The defense can also find a board certified expert who will look at the same record and the same circumstances and swear that there was no deviation from his/her definition of the standard of care or no causal relationship with the injury. Who is a medically unsophisticated jury to believe? The one

(Continued on page 339)

A Different Perspective

ERNEST H. NEIGHBOR, M.D., J.D., *Kansas City, Kansas*

“POLARIZED” . . . No better illustration of the word has ever existed than the relationship between physicians and attorneys. Most physicians are in complete agreement with Dick, the butcher, who says in Shakespeare’s *King Henry VI, Part Two*, “The first thing we do, let’s kill all the lawyers.” In the opposite vein, many attorneys share George Bernard Shaw’s opinion of physicians: In the preface to *The Doctor’s Dilemma*, he writes “Treat every death (following medical treatment) as a . . . murder by making it the subject of a reasonably conducted inquest; and execute the doctor if necessary by striking him off the register.”

In Kansas, this polarization has significantly increased within the past few months as both sides appear to be headed toward direct confrontation over proposed tort reform in cases of medical malpractice. The statements of leaders on both sides demonstrate a significant lack of understanding for the total issue. Physicians lack an understanding of the legal process, and attorneys are unconvinced that malpractice suits and awards are causing a damaging distortion in the health care delivery system. If medicine is to achieve success in the legislature, it would seem physicians should first educate themselves concerning the position of the other side, so that their proposals demonstrate an understanding of the problem and a desire to work toward the fairest possible solution. In order to understand the other side’s point of view, it is necessary to first examine the legal process and more particularly the role played by the plaintiff’s attorney.

The Plaintiff’s Attorney

Because of training and the realities of the legal process, the attorney has only one goal — to achieve the maximum benefit for his/her client. If representing the plaintiff, the attorney’s job is to obtain the greatest amount of money possible no matter how minor the underlying injury. If representing the defendant, s/he is working for a minimum award no matter how serious the injury and extensive the

negligence. This adversary nature of the legal process is illustrated in the selection of a jury. Attorneys readily admit that in choosing a jury they are not interested in jurors who will be “fair.” They want jurors who will favor their side. The fact that “fair” juries are generally produced is the result of one side counteracting the other.

Attorneys understand their role and the responsibility they owe to their clients. They owe no duty or responsibility to their opponents’ clients. Any responsibility to society is secondary. Their training teaches them that society will ultimately benefit if they just represent their clients to the best of their abilities. This duty to work for the maximum recovery for both present and future clients requires them to oppose any changes in the law that would reduce the potential recovery.

When medicine suggests that attorneys’ motives for fighting tort reform are nothing more than concern for their pocketbooks, it shows its own lack of understanding of the legal process. There have been many times when the positions taken by medicine on various issues for the benefit of patients had an indirect (and in some cases direct) effect on physician income. We need to recognize that the plaintiffs’ advocates have a right to resist changes that they see as being injurious to that portion of society that they represent.

This is certainly not to suggest that they are right and medicine is wrong — quite the contrary. The plaintiffs’ bar has been so successful that a situation now exists that is having an adverse effect on the health care delivery system. This is detrimental to society. The potential recovery of future plaintiffs may have to be somewhat curtailed in order to alleviate this problem. Physicians should recognize the right — indeed the duty — of attorneys to argue against tort reform.

As an illustration, consider the situation in the National Football League several years ago when all of the kickoffs were going through the end zone and runbacks were almost nil. No one criticized the kickers for developing their technique to the point that they could routinely boot the ball 65 plus yards. It was simply recognized that the state of the art had reached a point where the game was suffering, and the rules were changed. Does anyone remember any kickers supporting the rule change?

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Constitutionality

Before proposing legislation on tort reform, some understanding of constitutional law must be acquired. The United States Constitution does affect the changes that might be proposed. In the Fifth Amendment there is a phrase insuring to all citizens "due process and *equal protection under the law*." The Fourteenth Amendment to the same document requires that the states in the passage and administration of their individual laws must protect for their citizens all of the rights guaranteed by the Federal Constitution. One of the major lessons to come out of the 1960s and 1970s is that these amendments show one class of citizens cannot be treated differently from another.

By passing tort reform for only certain torts — *i.e.*, medical negligence — the legislature would be creating a special class of citizens and saying to them "We have special rules for you. If you were injured as a result of an auto accident or the structural failure of a building then you can recover under the old rules, but if you were injured as a result of the negligence of a health care provider then there are special rules and limitations that apply to the way you can go about recovering for your injuries." It is clear that such an approach has serious constitutional problems.

Occasionally, there is recognized such an overwhelming social need that special rules can be created. The most obvious example is the passage of the workers' compensation laws in the late 1800s which was justified by the plight of the injured worker. If medicine is to be successful in its efforts at tort reform, it must bear in mind the judicial review that such laws must ultimately face. There is no point in working hard to pass laws that will be overturned by the courts three or four years later. If changes in the rules governing medical malpractice are passed, a clear need for such special rules must be shown. On the other hand, if there is a need to make changes in the rules that govern the litigation of all forms of negligence cases — not just medical negligence — then this is the goal toward which physicians should be working. Medicine could be joined in this effort by the various organizations sharing its view of the need for tort reform. This type of legislation would rest on more solid constitutional footing.

It must also be borne in mind when considering the constitutionality of proposed legislation that our elected representatives must give considerable thought to the constitutional problems of the legislation that they are asked to support. There are explanations other than simple self interest to explain why legislators may not favor a particular piece of

legislation. Once again, the medical profession should consider all the possible factors that motivate our legislators. Physicians need to understand the constitutional problem and work with our legislators to achieve the tort reforms that will have the best chance of passing constitutional muster.

Pain and Suffering

The American people are among the most generous and caring individuals on earth. They become even more generous when the money is not coming from their own pockets. This fact plus the current state of the art in trial advocacy have pushed the awards for pain and suffering far beyond the limits that the system can tolerate. The evidentiary use of television videotapes to present "A day in the life of" the injured party to the jury combined with the skills of the "world class" attorney sends members of the jury into the jury room with sympathetic tears in their eyes. They are mad at the defendant, and ready to award the world to the injured plaintiff. The foreman of the jury in the Sally Firestone case (the most severely injured survivor of the Hyatt Hotel disaster — Award = \$15 million) stated to the press that the jury had wanted to put Ms Firestone in the position of never having to worry about a thing for the rest of her life. Clearly, this went far beyond the purpose of the tort system.

Unlimited awards for pain and suffering allow the jury to inflict punishment on the defendant without the plaintiff having to prove the need for punitive damages, and this also goes beyond the purpose of the tort system. Unnecessary pressure is put on the judiciary by these exorbitant awards when the defendant requests a reduction of an award that was clearly the result of emotion — sympathy, anger, or both.

A statute has been on the books for many years that limits the amount that can be recovered over and above actual monetary losses in suits involving death. Currently, that amount is \$100,000. Consider the amount of grief and pain caused to a wife and children from the sudden loss of a husband and father. If a dollar limit can be placed on this, it seems only logical that a limit can be placed on the amount of money that the jury can award to an injured party in a negligence case.

Collateral Source and Attorneys' Fees

With the almost universal availability of health insurance — either through employment or some government program — there would seem to be no justification for continuation of the collateral source rule which prevents the defendant from showing that some or all of the plaintiff's medical bills have been

paid by insurance. The plaintiff should be reimbursed only for out of pocket medical expenses. The collateral source rule provides the plaintiff with unjust enrichment and unnecessarily increases the cost of insurance. It should be revoked.

Much has been written concerning the amount of the award that attorneys take as their fee. A detailed discussion of the topic is not possible within the scope of this article. Clearly, if the attorney is actually taking 50 per cent of the multi-million dollar award, there is inequity in the system. However, if there were a limit on the award for pain and suffering and if the collateral source rule were overturned, the contingency fee would probably have a more equitable relationship to the work involved and the risk taken by the attorney. Physicians dislike having their fees fixed or limited, and attorneys are no different. If the jury awards can be made more reasonable by the adoption of the above-mentioned reforms, there is probably no reason for medicine to concern itself with the contingency fee question.

Continuing Education in Risk Management

Seminars in risk management can have a positive effect on the malpractice problem. There are patterns in the way medicine is practiced that are conducive to negligent occurrences. Other patterns create an environment that lessens the chance of suit even when a less than hoped for result has occurred. Still other patterns of practice can significantly influence whether a suit, once filed, can be successfully defended or not. By making the physician more knowledgeable in these matters, the malpractice climate can be improved. There is only one problem — physicians who would voluntarily attend such a seminar are generally the ones who need the information the least. A great many physicians would benefit greatly from being exposed to such a program at regular intervals. If most physicians were provided with a better understanding of risk management, perhaps they could influence the few whose patterns of practice could not be changed by the educational process.

As part of its program for improvement of the medical malpractice situation, organized medicine should propose that a portion of the continuing education requirement for licensure be done in risk management seminars (for instance, 10%). Medicine will be asking the legislature for changes in the legal system, and it seems only appropriate that physicians demonstrate their willingness to make

changes in the medical profession to improve the situation.

Conclusion

This article is by no means meant to be an in depth discussion of the medical malpractice problem. Rather, it is meant to suggest a different way of looking at the legal profession, a few of the more obvious ways in which the tort system could be changed, and a proposal that, if passed, would demonstrate physicians' willingness to improve the situation. There is much more that needs to be done in the way of thought and discussion. It is to be hoped that education about the problem will foster thought, discussion, and proposals that are based on solid reasoning rather than emotion.

Medical Witness

(Continued from page 336)

who looks and acts most like Marcus Welby? When experts cannot agree among themselves, what chance does the layperson have of rendering a fair decision?

The recommendation made here, which is not unique or revolutionary, speaks equitably to the causes of both plaintiff and defendant. Justice would best be served if expert medical witnesses or a panel of such experts could be *amicus curiae*, advisors to, friends of the court. Such witnesses would inform the judge and jury in the interests of communication and understanding rather than partisanship and advocacy. Legal minds must determine the details of implementation, but the advantages of such a system would be numerous. The most important benefit, however, would be the restoration of scientific objectivity when advising those who must judge an inexact science as well as those physicians who seek to heal through the application of that science.

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The Search for Permanent Resolution

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THE KANSAS Medical Society, through its Professional Liability Committee, will propose a package of legislation for tort reform to the 1985 Kansas Legislature. Foremost among their proposals will be the limitation of awards for pain and suffering as well as a limit on attorneys' fees. Should they pass, there is no doubt that these two items will restrain the ever-increasing cost of liability insurance premiums. Although the immediate effect will be to slow the rising cost of litigation, it is possible that in the long run these measures will prove to be only stop-gap measures, and during the next few years insurance premiums may again increase rapidly to a prohibitive level. For this reason, we should look beyond 1985 to find a more permanent solution to this problem that has plagued us for the past 20 years.

The need for change in the present system has been recognized by many people other than physicians. In 1982, Chief Justice of the Supreme Court Warren E. Burger addressed the issue in his annual State of the Judiciary address to the American Bar Association. His speech was titled, "There Must Be a Better Way." In addition, the prestigious McGill Commission has stated:

. . . Inasmuch as the present tort law liability insurance system for medical malpractice will eventually break down and costs have and will continue to rise to unacceptable levels, fundamental reform of the present tort law liability insurance system should be undertaken. The overriding concern should be to create a system of compensation for adverse medical outcome resulting from medical treatment whether or not caused by negligence . . .

Even the federal government's own interagency task force on product liability, which is being hit just as hard as medical liability, in a report in 1977 emphasizes "growing appreciation of the need to explore more carefully the utilization of the 'no fault' compensation system for product liability."

Two other possible methods have been proposed. One would require patients to purchase a type of medical/accident insurance when they are admitted to the hospital. This would then cover them for any time lost from work or disability caused by complications from a surgical or medical treatment. Another proposal is the "no fault" system, some-

what similar to the workers' compensation system which now exists in all 50 states.

There are many in both the legal and the medical professions who dissent. They argue that instituting a "no fault" system would create more claims and in the long run would prove more expensive than the present system. However, statistics show this is not true in the case of automobile "no fault" insurance. Attorneys' strongest argument against instituting such a system, of course, would be that it would be unconstitutional through denial of a person's right to his/her day in court. However, the precedent has been set with workers' compensation and "no fault" automobile insurance. In addition, under certain circumstances, a patient/plaintiff could still have his/her day in court. The attorneys' fear, of course, is of an adverse effect on their incomes. However, since plaintiffs would still have the right to be represented by legal counsel, the majority of trial lawyers would be unlikely to suffer serious loss.

A "no fault" system that many envision would compensate plaintiffs only for economic loss and for no other reason; *i.e.*, there would be no compensation for pain and suffering, for loss of consortium, or for any other type of non-economic loss. Under this system, patients injured while undergoing medical treatment would be compensated quickly and fairly, and would receive more of the premium dollar. Under the present system, the patient/plaintiff receives only about 25 per cent of the money paid for insurance premiums, and often s/he has to wait from two to ten years before receiving any of it. While more people may be compensated under this system, it probably is fair that they should be. Most physicians are sympathetic and would not oppose compensation for patients who sustain a real economic loss secondary to a complication. This does not mean, of course, that every patient would be compensated because his/her condition deteriorated despite adequate medical treatment, but it should not be difficult to set up a medical compensation board similar to a workers' compensation board to determine if and how much compensation each person should receive.

Because it will probably be several years before such a system can be instituted or even justified in the state, physicians must continue to work with the Medical Society through the state legislature for

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Law Students/Liability/Compensation

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FAULT . . . Liability . . . Adversary . . . Litigation. Law students are intimately acquainted with such words. They are less familiar with words such as negotiation, settlement, mediation, and nonjudicial arbitration. Law school graduates carry a “fault” repertoire to their eventual practice. Consequently, the “fault” frame of reference pervades our society’s legal and judicial systems.

Why are law students so familiar with fault and similar terms? They are the products of their education. Law schools emphasize fault and adversarial approaches to dispute resolution. Teaching methodologies, testing, and grading contribute to an obsession with fault and adversarial relationships, and hiring practices of new attorneys reinforce the fault and adversarial frame of reference.

Beginning in the 1800s at Harvard Law School, the case system was established as the basic method of teaching law to replace the lecture system — professorial statements of law in essentially nonpersonal terms. The case system is essentially the study of the highest court decisions with the actual parties and their legal problems being handled and decided. Hence, a casebook in contracts might well present a case decided by the Kansas Supreme Court entitled *Smith vs Jones*, and affirming the lower court decision awarding damages to Smith because Jones breached the contract and was therefore *liable* to Smith in damages.

Current teaching methodologies require law students to read thousands of court cases. Within the cases, the facts describe two stories. The courts apply to such facts a rule of law. The rule of law is derived from previous case law and statutes. From this application of a rule of law to the facts, one story prevails. The court grants favor to the winner’s requests and renders fault and liability to the loser. The court cases evidence the adversarial system at work.

The Anglo-American system of creating law is based upon expansion of rights with a concomitant expansion of liability based upon fault. For example, the California Supreme Court has recently expanded rights to a party in a contract relationship by

holding that in certain cases damages may be based upon a tort theory of liability where a party has willfully breached a contract and then seeks to shield itself from liability by denying, in bad faith and without probable cause, that the contract exists.

The reading of such cases can distort the student’s perceptions of the role of the law profession. The cases render an impression to the student that contracts are ordinarily breached. The student discovers that wills appear to be under constant legal attack. Students can conclude that all attorneys spend the majority of their time litigating such disputes.

Justice appears to the students to rest on finding fault and liability of one of the disputing parties. After reading volumes of case law, the students can infer that adversarial parties must battle to reach truth and justice. They can conclude that truth and justice require only one victor.

The students learn that an attorney is to be a zealous advocate for his/her client. The attorney ordinarily is to argue the client’s point of view — the client’s truth. The attorney’s job as an advocate is to make sure the client is the victor in the dispute.

The fault and adversarial approach to law does not always render truth and justice. The approach essentially renders a victor. Negotiation and settlement, mediation, nonjudicial arbitration and workers’ compensation courses offer an alternative route to truth and justice for all. However, law students are not required to take such courses for graduation. Many students leave law school without becoming familiar with negotiation, settlement, mediation, and compensation without fault. Therefore, the current teaching methodologies that stress case law study perpetuate the fault and adversarial approaches.

Law school testing contributes further to the fault and adversarial approaches to law. For the majority of students, course grades are based on a semester-end examination. The examination is generally an essay test that lasts for several hours. The students are given complex fact situations. Often they are to respond with the liabilities of the parties in question. Possible negotiation and settlement between the parties is not a consideration. Therefore, the students spend the entire semester preparing the best answers regarding the fault and liability of the parties. Such testing further imprints the fault and adversarial approach in the students’ minds.

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Law school grading and current hiring practices also are factors in strengthening students' adversarial attitudes. Preparing for the best test answers stems from the students' desire and need to excel above fellow classmates. With the overpopulation of attorneys and law students, superior grades are extremely critical for the student's future. Many top firms refuse to interview senior students who are not in the top 10 or 20 per cent of their class. Such fierce competition for grades and jobs creates an adversarial environment among the students. Under such conditions, a win-all attitude may supersede a best-for-all frame of reference.

As a result, law students quite naturally leave school with a firm attachment to the terms fault, liability, adversary, and litigation. Students have been prepared to view their profession in such terms, and have learned to excel within such contexts. Negotiation, settlement, mediation, and compensation without fault may not be a part of the student's repertoire. Obviously, practicing attorneys find a high level of comfort with the familiar fault and adversarial approach. Therefore, the fault frame of reference continues to be the legal profession's response to many societal problems, as contrasted to the compensation system based upon a foundation other than fault.

The concept of recovery of rights owed based upon liability or fault has been a part of our legal system since the earliest days of American and English law. In those very early days the English crown wanted rights to be taken care of by those at fault so there would be "no clamor for want of justice," and indeed fault was equated with quasi-crime. But in the middle 1800s in Europe came the beginnings of compensation to an injured person without simultaneously finding fault. The basic theory of these statutes was simply this: that in certain areas, and specifically in injuries to workers while working, it was important to both the individual and to society that income be continued after the injury and that the need to find fault was overridden by the need to compensate. In the civil law countries in Europe, such as Germany and France, where law is not judge-made, this new concept was embodied in statutes. In England, where the law was largely judge-made, the concept was developed by some judge-made changes to compensation without fault, but was largely developed by statutes making changes or erosions in the fault concept. In England and in the United States in the 1800s these changes were called "employer liability laws" — so called because of the theory that the blood of the worker was part of the cost of producing the product by the employer and

such cost would be passed on to the consumer as part of the price paid by the buyers. In England, with no written constitution worrying Parliament, these statutes that affected fault included such concepts as contributory negligence, the fellow-servant rule, and assumption of risk (all great defenses of the employer when sued by an injured employee). They nibbled at the tort liability concept, and were soon followed by the adoption of a workers' compensation statute which abandoned the theories of negligence and tort and set up the compensation without fault system.

In the United States, the bugaboo of "unconstitutionality" has always worried legislators — both in Congress and in each state. Basically, this worry manifests itself in a free-floating anxiety on the part of legislators, attorneys, professors, and students of law — the anxiety being that any change in the basic concept of fault may deny due process under the U.S. Constitution, or a state constitution where the change is made, or both. In addition to deprivation of due process, other anxieties involve equal protection of the law and an almost endless array of similar problems, any one of which can lead a court to state that the legislature has acted in violation of a constitution — which power courts have exercised in the United States since *Marbury vs Madison* in 5 U.S. (1 Cranch) 137 (1803).

Whether Justice Marshall was right or wrong in saying courts in the United States had this power, the concept is no longer debated seriously. In the area of railroads, Congress has never left the "employer's liability" concept. But, all states have changed the entire ballgame and passed workers' compensation statutes, such as Kansas in KSA 44-501 et seq., which statutes set up an entirely new concept of compensation based upon an obligation other than tort. Kansas was one of the first in this area, passing the law in 1911. It has been amended many times, but the basic concept remains the same. It has been held constitutional in Kansas and similar statutes are in place in all states. The free-floating anxiety in this area of the law has been quelled.

What are the basic concepts of workers' compensation that make it constitutional? No individual has an *absolute* right to factors leading to due process and equal protection under the law. The sovereign authority of the state empowers it to modify its rules. Therefore, a state can replace a fault liability system with workers' compensation, which balances employees' and employers' rights. Employees relinquish their right to sue employers for work-related injuries. However, employees are ordinarily entitled to moderate compensation in all

work-related injuries. Employees receive a more immediate compensation as opposed to possible damages in a costly, slow trial proceeding. Employers relinquish their defenses to fault. They are basically responsible for work-related injuries regardless of fault. However, the employer's liability is substantially limited. Employees' injuries are foreseeable in modern enterprises. Under workers' compensation, employers do not face sporadic and expensive lawsuits. The employer's insurance cost under workers' compensation is a predictable business expense, and is an equitable compromise for employers and employees.

The Kansas Workers' Compensation Act, KSA 44-501 et seq., continues the theme of balancing rights. The act applies to all employments where the employer's gross annual payroll is greater than \$10,000. The act excludes railroad and agricultural enterprises. However, excepted enterprises may elect to participate under the act. Widespread participation in the act promotes the program's goals and continuance.

The Kansas act provides compensation to the injured employee in conformity with its provisions. The injured employee may receive weekly benefits for injuries, plus medical and rehabilitation benefits. Property damage is not compensable. The dependents of a deceased worker may qualify for death benefits. Under workers' compensation, disability benefits are viewed as a replacement of wages. The disability benefits maintain some income for the worker and the family, as well as the worker's spending impact on the economy. In Kansas, the disabled worker receives a percentage of his/her average weekly wage. Medical benefits to the injured worker are unlimited in Kansas. Kansas is a leading state in the provision of rehabilitation benefits to applicable workers. Death benefits provide burial costs, and compensation also is payable to statutory dependents who rely upon the deceased worker's earnings. Compensation benefits the worker, the family, and society.

The Kansas act is a scheme of social legislation that aids those suffering a "personal injury by accident arising out of and in the course of employment." "Arising out of" refers to the causal connection between the accidental injury and the employment. "In the course of" alludes to locating the injured worker at the place of service. Questions of the act's coverage are infrequent. Kansas courts have chosen a liberal interpretation of the act to promote the legislative intent of protecting injured workers.

Workers' compensation rights are contingent

upon an employer-employee relationship grounded upon an express or implicit contract. The employer's fault is not at issue. However, the act excludes compensation for employee injuries under certain circumstances. Compensation is excluded when workers are disabled for less than a week. Workers' injuries resulting from their deliberate intention to cause such injuries are excluded. Compensation is not permitted for workers' willful failure to use safety devices, statutorily required or voluntarily furnished by the employer. Injuries resulting from the worker's intoxication or usage of certain drugs also are excluded. The act also excludes most coronary, coronary-artery disease, or cerebrovascular injuries from compensation, although it does cover such conditions if it is shown that the work exertion necessary to precipitate the injury exceeded the worker's usual work. The focus on the employer/employee relationship promotes the balancing of rights.

Questions do arise as to the compensation due under workers' compensation. Injured employees are categorized to aid in the calculation of compensation. Injured workers are classified as temporary partial, temporary total, permanent partial, and permanent total. Hospitalized workers are ordinarily termed temporarily totally disabled. Permanent partial usually refers to scheduled injuries such as the compensation amount for the loss of a thumb. Ordinarily, permanent total disability refers to injuries that affect the functioning of the whole body. The loss of both eyes could be considered as a permanent total disability. Such categorizations enter into the complex calculations of compensation entitlement.

The Kansas administrative procedure for obtaining and enforcing compensation rights begins with the occurrence to the worker and subsequent notice. The employee gives notice of the accident to the employer and to the workers' compensation director. The worker's notice of the occurrence to the employer is waivable. Settlement between the employee and the employer/insurance carrier may occur at this time. If settlement does not occur, the worker makes a compensation claim to the employer and workers' compensation. The worker may apply for immediate compensation. If immediate compensation is granted wrongfully, the employer is entitled to reimbursement from a state fund. Following the claim, an administrative law judge conducts a hearing. Compensation can be awarded as a lump sum or on a periodic basis. After the judge's ruling, the workers' compensation director gives express or implicit approval. If no court appeal is made within

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Medical Malpractice Legislation: Kansas

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THE UNITED States is suffering from a medical malpractice crisis. The crisis is generally characterized by rapidly rising premium rates for medical malpractice insurance and actual or threatened loss of insurance coverage. It first surfaced in the late 1960s and early 1970s when dramatic increases in malpractice claims and awards forced insurers who had previously written policies covering medical malpractice to withdraw from the market. Insurance companies that continued to provide coverage drastically increased their rates and covered fewer risks in order to realize a profit. Physicians unable to afford the escalated premiums feared that they would be left uninsured.

Legislators across the nation believed the difficulty of obtaining affordable malpractice insurance would threaten the availability and quality of health care. In response to this concern, from 1975 to 1977 virtually all states enacted or considered medical malpractice legislation.

Legislative action taken by the states in response to the crisis can generally be divided into two categories. The first involves legislation concerned with the availability of medical malpractice insurance. Legislation creating joint underwriting associations was enacted by many states to resolve the dilemma of insurance availability. Although the structure of the underwriting association varies between states, the main purpose of the underwriting programs is to provide malpractice coverage at affordable rates.

The second category entails statutory provisions that alter the substantive and procedural rules utilized in malpractice cases. This class of legislation is designed to decrease the liability of health care providers and thereby reduce the risk of coverage to insurers so that malpractice insurance can be made available at affordable rates. These legislative measures include statutory revisions in licensing requirements of physicians, institution of screening and arbitration panels, statutorily imposed limitations on

the amount a plaintiff can recover in malpractice cases, alteration or elimination of the collateral source rule, imposition of an attorney contingent fee schedule, and reduction of the statute of limitations period for medical malpractice actions.

Numerous factors have been cited as contributing to the initial malpractice crisis. The increasing amount of literature on new medicines available to the public which inform the public on the proper administration of medicines, and the current trend toward the specialization of physicians which results in decreased physician-client rapport and less frequent patient contact are two frequently cited factors. In addition, there seems to be a general public attitude that anytime a person is injured a lawsuit should be instituted. Other factors cited include unrealistic recovery expectations of patients, the unpredictability associated with jury awards, and an increased exposure to medical care.

The Health Care Stabilization Act

The Kansas reaction to the medical malpractice crisis has been primarily in the area of insurance availability. In 1976, Kansas enacted the Health Care Stabilization Act, which is comprised of three tiers of legislation designed to ensure the availability of malpractice insurance. Under the first tier, health care providers are statutorily required to maintain medical malpractice insurance. The second tier formulates a joint underwriting association, and the third tier establishes the Health Care Stabilization Fund.

Initial Provisions

Under the provisions of the Kansas Health Care Stabilization Act, all health care providers are required to maintain medical malpractice insurance. The Act formerly required every health care provider operating within the State of Kansas to carry a minimum basic coverage of \$100,000 per occurrence, subject to a \$300,000 aggregate for all claims made during the policy period. Amendments by the 1984 legislature included an increase in the minimum basic coverage to \$200,000 per occurrence with a \$600,000 aggregate. Resident and nonresident providers who fail to maintain the basic coverage are prohibited from rendering professional services in Kansas.

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Ms. Wedel has updated and revised the material for publication here.

The constitutionality of the Kansas mandatory insurance program was challenged in 1978 in *State ex rel. Schneider v. Liggett*. In *Liggett*, a physician who was enjoined from practicing medicine for failing to obtain medical malpractice insurance, alleged the statute requiring physicians to maintain insurance violated the due process and equal protection clauses of the 14th amendment. The physician argued that he had a vested right to practice medicine without maintaining insurance because he had been licensed and qualified to practice before the statute was enacted. He contended that additional requirements imposed by the state that took away this vested right would violate due process unless the additional requirement was directly related to a person's ability to practice medicine. The court did not agree.

The Kansas Supreme Court found the enactment of a mandatory insurance program did not violate due process because the legislation has a rational relation to the health and welfare of citizens of the state. Requiring physicians to maintain malpractice insurance protects citizens harmed as a result of malpractice and assists in assuring the continued availability of medical care.

The physician's constitutional attack predicated on equal protection was also rejected by the court, which ruled that the practice of medicine does not involve a fundamental right. As such, statutes regulating the medical profession will be set aside only when it is shown that the statute is wholly irrelevant to the state's objective in passing the regulation. Although the court recognized that the legislation treated high risk surgeons the same as low risk surgeons and excluded other professionals such as nurses, dentists and attorneys, the court found these discrepancies insufficient to declare the statute unconstitutional.

Another significant feature of the Kansas Health Care Stabilization Act is its formation of a joint underwriting system designed to assure the availability of malpractice insurance. The Act directs all insurance and rating organizations to cooperate in insuring providers who, although in good faith are entitled to insurance, are unable to procure it through ordinary means. The underwriting plan directs these companies to reinsure risks of a designated serving carrier selected from the insurance companies. Unlike other types of joint underwriting association schemes, the insurer is not assigned individual physicians for which he or she must provide insurance. Instead, the servicing carrier reinsures the other insurance companies participating in the plan.

The plan is operated so that the insurers neither

make nor lose money. If the expenses of the joint underwriting system exceed the profits, the insurance companies are reimbursed for their losses. Reimbursement is made out of the Health Care Stabilization Fund which is supported by surcharges paid by health care providers. When profits exceed losses, the insurance companies must pay the surplusage into the fund. In 1982, for the first time, the underwriting losses exceeded the profits by \$1 million. Thus, the fund was liable for this amount at the end of the 1982 fiscal year.

One of the major features of the Kansas Health Care Stabilization Act is the creation of the Health Care Stabilization Fund. The fund, which is administered by the insurance commissioner, was created to provide coverage for judgments or settlements exceeding the basic coverage policy limits.

The protection of the fund extends to all providers who are residents of Kansas, regardless of whether the act of malpractice occurred in Kansas or some other state. Nonresident providers are protected by the fund for malpractice liability that occurs within the state as long as the nonresident has complied with the basic coverage requirements.

A provider who originally complied with the mandatory basic coverage provisions but is no longer rendering professional services in the state is labeled an inactive health care provider. Inactive providers, such as those who retire or move from the state, will no longer carry a basic coverage policy. Yet, malpractice claims may be filed against them based on an act of malpractice that occurred when the provider was still rendering medical services as a Kansas practitioner. Since inactive providers have no policies to cover claims filed against them, the fund is obligated to pay damages from the first dollar rather than the amount exceeding the basic coverage requirements.

The fund is supported by a surcharge levied by the commissioner. The amount of the surcharge is a percentage of the premiums that must be paid by the provider in order to maintain the basic coverage requirements. If the provider is self-insured, the surcharge is a certain percentage of the premiums the health care provider would have had to pay to maintain the basic coverage requirements if he or she were not self-insured.

Although the commissioner selects the applicable percentage rate for the surcharge, the legislature has statutorily prescribed guidelines the commissioner must follow in determining how awards against the fund should be administered. In 1983 and 1984, the legislature made changes to the original guidelines given in 1976 when the Act was first adopted. The

changes will affect the administration of the fund during the fiscal years of 1984 and 1985. The statistical effect of the 1983 and 1984 changes have not yet been ascertained. Consequently, it is necessary to examine the legislation as it existed prior to the recent legislative changes and the fund's effectiveness under the original act before addressing the 1983 and 1984 changes.

Prior to the legislative changes made in 1983 and 1984, the commissioner was directed to charge a rate calculated to accumulate \$10 million by the year 1990. Until the fund had accumulated \$5 million, the surcharge could not be less than 40 per cent. If the fund exceeded \$10 million, the commissioner was directed to reduce the surcharge so the fund could be maintained at the \$10 million level. At no time could the surcharge rate exceed 65 per cent. Providers who complied with the act for the first time were required to pay a minimum surcharge of 25 per cent.

In order to reach a \$10 million balance in the fund, the commissioner levied a 45 per cent surcharge in 1977 and 1978 followed by a 40 per cent surcharge in 1979. In 1980, the surcharge was 15 per cent. No general surcharge was levied in fiscal years 1981-83 because in 1981 the fund had reached a level of approximately \$14 million. Because the balance was in excess of \$10 million, the commissioner was prohibited from imposing a surcharge.

The large cash balance in the fund during the years when no surcharge was levied is somewhat misleading. Under the 1976 legislation, the fund could disburse only \$300,000 per year, per claim. Any claim awarded against the fund that exceeded \$300,000 had to be paid by installment payments of \$300,000 per year. Consequently, the fund was obligated to make future payments on claims greater than \$300,000 that were awarded against the fund. These future payments were not reflected by the cash balance of the fund. Yet, prior to the 1983 changes, the cash balance for a fiscal year was the mechanism for determining the surcharge rate. Thus, even though the balance of the fund at the end of 1982 was about \$12.5 million and no surcharge could be levied, future liabilities against the fund were substantial. At the end of the 1983 fiscal year the Insurance Commissioner's office estimated that future obligations totaled more than \$2.7 million.

Few claims requiring future obligations against the fund occurred during the first four years of the fund's existence. From 1977 to 1980, only 3 per cent of all claims that resulted in an award against the fund were paid. Although approximately 151 claims were filed against the fund during all three years

combined, only four resulted in an award. The total amount claimed against the fund in each of those years never exceeded the \$300,000 maximum amount that could be paid out each year for any one claim.

Since 1980, the number of claims and awards against the fund has climbed dramatically. In 1981, the number of claims filed increased from the 82 claims filed in 1980, to 100 claims — a 22 per cent increase. The eight cases that were settled during 1980 were settled at a cost of \$1.7 million. Nevertheless, the fund at the end of the 1981 fiscal year maintained a \$13 million balance.

The drastic increase in the fund's liability did not end with the \$1.7 million settlement costs of 1981. Statistics show claims awarded against the fund during 1982 at approximately \$3 million, the result of 24 claims settled in 1982. A total of 124 claims were filed in 1982. This constituted a 24-case increase over the 100 filed in 1981. Thirty-two of the cases filed in 1981 were closed, either because the claims were settled or the action was dismissed. Sixty-six cases were closed during 1982, which left 243 active cases pending at the end of fiscal year 1982.

The picture looked no better in 1983. More than 15 new cases were filed, an increase of at least 30 per cent over 1982, and cases pending grew to a total of 288 cases. Twenty-five of them were settled in 1983 at a cost of \$6.5 million.

Cumulative figures indicate that a dim future may be in store for the fund's financial stability. A total of 531 claims have been filed against the fund since its inception. While 243 of these cases had been closed by the end of the 1983 fiscal year, 288 remained active. As of 1983 the average claim paid against the fund was estimated at \$191,712.

If the escalation of the first seven years continues, the fund may soon become unable to fulfill its obligations. Its balance at the end of fiscal year 1983 was about \$8.3 million. However, future obligations through 1988 were estimated to be \$2.7 million.

Recent Legislation

In 1983 and 1984, the Kansas legislature responded to the fund's problems by amending the original act with changes designed to maintain its solvency. The legislature repealed the provision that precluded the commissioner from levying a surcharge if the fund had a cash balance of \$10 million. The commissioner can now levy an annual surcharge against providers regardless of the fund balance. The surcharge is to be an amount deemed sufficient by the commissioner to fund anticipated claims.

The legislature also changed the provision limit-

ing the amount the fund can pay on any claim to \$300,000 per year. Under the new law, the fund can pay either \$300,000 per claim or 10 per cent of the claim, whichever is greater. This change is especially important in cases involving large claims. When million-dollar awards are made, the \$300,000 per year limitation may result in long-term future obligations. The ability to pay 10 per cent of the claim per year ensures the claim will be liquidated in a ten-year period.

Perhaps the most significant legislative enactment is the 1984 amendment that limits the fund's liability to \$3 million for any one judgment or settlement, subject to a \$6 million aggregate of all claims made against a health care provider in any one fiscal year. In light of recent malpractice verdicts in the million-dollar range, the \$3 million cap may be the best means of protecting the fund's long-term solvency.

Peer Review

Although legislation to increase the basic coverage requirements and limit the fund's liability assists in controlling the fund's financial stability, it does not reduce the number of medical malpractice claims filed against physicians or control the quality of medical care. Some suggest that effective peer review programs may help to achieve these goals.

Kansas has established a State Board of Healing Arts to govern the licensing of medical practitioners. As of 1978, all practitioners who request license renewal are required to substantiate completion of a continuing education program. The board has the authority to revoke, suspend, or limit a license if a practitioner acts in an unprofessional manner. In 1983, the legislature added a new provision to the Healing Arts Act which requires physicians to report information concerning the violation of the Healing Arts Act. Any person who is licensed to practice the healing arts is required to report violations by other licensed health care providers.

Although the Kansas peer review system received a major setback in a recent Kansas Supreme Court decision, legislative action has been taken to revitalize and improve peer review programs. In a 1983 decision, *Wesley Medical Center v. Clark*, the Kansas Supreme Court allowed the plaintiff in a malpractice suit to discover documents and records made by a medical staff review committee. The committee was responsible for the evaluation of physicians working in the hospital. Legislators and medical professionals were extremely disturbed by the court's decision. They feared that permitting plaintiffs access to medical peer review records would deter health care organizations from evaluat-

ing their own physicians because of the potential wide exposure of liability for both the evaluated physician and the facility. In addition, they feared that the evaluated physician would not be as willing to provide information regarding his or her own conduct if records were not kept confidential.

In response to these concerns, the 1984 legislature passed legislation that makes information obtained by a peer review committee privileged and nondiscoverable. The legislation also includes provisions that create a board of governors authorized to assist in providing appropriate disciplinary measures for unprofessional medical conduct. The board of governors is comprised of 12 health care providers and the insurance commissioner. If the board of governors determines by a majority vote that a health care provider presents a material risk of significant liability to the fund, the governors, after notice and an opportunity for a hearing, may exclude the provider from the fund's protection.

Another section of the bill provides for the appointment of a disciplinary counsel to investigate matters involving professional incompetency. At the conclusion of the investigation of the conduct of a health care provider, unless the complaint is determined to be unfounded, the disciplinary counsel presents matters involving professional incompetency or unprofessional conduct to a review committee. The review committee is comprised of three members appointed by the State Board of Healing Arts. If the review committee recommends the matter for hearing, the disciplinary counsel is directed to file a petition with the State Board of Healing Arts for revocation or suspension of the health care provider's license.

The creation of the board of governors and the disciplinary counsel position give the medical profession an opportunity to correct the malpractice crisis within their own field. With this legislation, the medical community will be able to increase the quality of medical care through stricter disciplinary measures for health care providers.

The Statute of Limitations

Many states have reacted to the malpractice crisis with legislation that shortens the time period for filing a medical malpractice action. By shortening the statute of limitations, legislators hoped to reduce the volume of malpractice litigation and thereby decrease malpractice insurance premiums.

Medical malpractice actions can involve complex medical issues that tend to make the time period between the negligent act or omission and final resolution of the claim longer than in other tort actions.

This phenomenon, known as the "long-tail" effect, often makes it difficult for plaintiffs to file suit within the statute of limitations period.

Many courts, sympathetic to plaintiffs barred from suit by the statute of limitations, created judicial exceptions to the statutes to extend the time period in medical malpractice actions. One of these exceptions, known as the "discovery rule," states that the statute of limitations begins running only after the plaintiff discovers, or through reasonable diligence could discover, that his or her injury was caused by the physician's negligent act or omission. The rule prevents a plaintiff from being unfairly deprived of a cause of action before the harmful effects of the physician's negligence becomes manifest.

A second court-created exception to the statute of limitations is the "continuous treatment" rule. Under the continuous treatment rule, the statute of limitations does not begin to run as long as the plaintiff is still under treatment for the injury. The continuous treatment requires that the treatment be performed by the same physician, associate, or in the same hospital. The rule gives the physician an opportunity to correct any mistake or misdiagnosis before the harm is manifested. In addition, it recognizes that it is unlikely a plaintiff will file suit against a physician while he or she is still under that physician's care.

The judicial application of these rules allowed patients to file claims many years after the occurrence of the physician's negligent act or omission. This deviation from strict adherence to the statute of limitations restrictions increased the risk exposure for malpractice insurers. As a result, insurance carriers contended they could not accurately predict how far in the future their liability would extend for each policy period.

To combat the problem, Kansas, along with other states, enacted legislation that sets inflexible statute of limitation periods so that malpractice insurers can more accurately predict their annual liability. In Kansas, an action arising out of the "rendering of or the failure to render professional services" must be filed within two years from the incident. If the cause of action is not ascertainable within this two-year period, the cause of action can be filed up to four years after the negligent act or omission of the physician. In no event can an action be filed after this four-year period.

An intriguing feature of the Kansas statute of limitations is its elimination of the fraudulent concealment rule. The fraudulent concealment rule provides that a physician cannot use the statute of

limitations as a defense when he or she deliberately conceals facts that would inform the plaintiff of a cause of action for malpractice. Through the elimination of the fraudulent concealment rule, the Kansas legislature has technically made it possible for a physician to escape liability by preventing the plaintiff from discovering the malpractice for a four-year period. However, the physician may nonetheless face liability for fraudulent concealment based on the equitable theory of estoppel which prevents a person from benefiting from his or her own wrongful conduct.

In 1981, in *Stephens v. Snyder Clinic Association*, the plaintiff contended that shortening the statute of limitations violated equal protection because it created an arbitrary and discriminatory classification. The plaintiff argued that the statutory classification was unreasonable because it provided for a four-year limitation for physicians, yet allowed a ten-year limitation period for other tortfeasors. The court rejected the plaintiff's constitutional claim holding that the classification distinguishing health care providers was reasonable because it was a legislative attempt to assure continued quality health care in Kansas.

Although the Kansas statute assists malpractice insurers in predicting their liability, it may not substantially affect the number of malpractice injuries filed each year. Studies indicate that more than one-half of malpractice injuries are recognized within one month after the negligent act, and more than 90 per cent of injuries attributable to medical malpractice are discovered within one year.

The Collateral Source Rule

Large damage awards in medical malpractice actions have been cited as a contributing cause of the medical malpractice crisis. A viable means of reducing damage awards in malpractice actions is through the abolition of the collateral source rule. The collateral source rule is a common law tort concept which prohibits the introduction into evidence of other sources that compensate the plaintiff for the same injury. This includes insurance.

The rule plays an important role in medical malpractice actions because the injured party frequently has maintained some type of insurance. Under the collateral source rule, a plaintiff is entitled to make full recovery against a health care provider even though the plaintiff is fully compensated by insurance proceeds. If evidence of other compensation, such as health insurance, could be submitted to the jury, it is likely that the verdict would reflect only the

damages exceeding the plaintiff's insurance policy limits.

Proponents of the collateral source rule argue that admitting into evidence at trial of the plaintiff's insurance coverage penalizes the plaintiff for having the foresight to maintain an insurance policy. This result has been criticized as discouraging the purchase of insurance. Proponents of the collateral source rule fear that reduction of the actual damages assessed against defendants by admitting evidence of collateral compensation will diminish the deterrent effect of liability.

Although these justifications have some merit, in medical malpractice actions they are not as persuasive. The main purpose of a medical malpractice suit is to compensate the victim for any harm resulting from the injury. When compensation is allowed both from the plaintiff's insurance and the jury verdict, the plaintiff is over-compensated for the injury. In addition, it is unrealistic to presume that the abolition of the collateral source rule would discourage the purchase of insurance because most insurance that covers injuries sustained from medical malpractice injuries also covers a wide range of other events. For example, health, life, and disability insurance cover conditions created by natural causes as well as conditions caused by the negligent acts of other tortfeasors. These other insurable interests will encourage plaintiffs to continue their purchase of insurance. In addition, if the defendant is required to reimburse the prevailing plaintiff for all premiums the plaintiff paid in order to maintain the insurance policy, the plaintiff may not be deterred from purchasing insurance.

Some states have enacted legislation entirely eliminating the collateral source rule in medical malpractice actions. Other states have only modified the rule.

In Kansas, the legislature chose to limit the collateral source rule in malpractice actions rather than totally abolish it. Under the statute, which has since been held unconstitutional, evidence of reimbursement or indemnification received by the injured party was admissible at trial unless the source was insurance or services partially paid for by an employer. In essence, the statute permitted evidence of services or payments made by public sources and benefits from gratuitous sources to be admitted to trial. The statute applied only to medical malpractice actions.

Although that statute was held unconstitutional, the court indicated that total elimination of the collateral source rule in medical malpractice cases may be constitutional. Total abrogation of the rule would

alleviate any unconstitutional distinction between insurance sources and gratuitous sources. Other jurisdictions have held such statutes constitutional.

Even though not as much money would be recovered by each individual plaintiff if the rule is totally abolished, the public at large may reap the benefits. Rising costs of medical malpractice insurance necessarily mean rising prices for medical treatment to cover insurance expenses. If damage awards continue to climb at the current rate, medical costs to the public will continue to escalate.

Sliding Scale Contingent Fee Schedule

In most medical malpractice cases, the plaintiff's attorney is compensated on a contingent fee basis by which the attorney is paid a certain percentage of the money awarded to the plaintiff — *i.e.*, the attorney receives compensation only if he or she wins the case. If the attorney loses the case, the plaintiff suffers little or no monetary loss.

Critics of the system argue that the contingency fee method of compensation encourages plaintiffs to file suits that otherwise would not be filed and that the cost of defending such suits contributes to the malpractice crisis by fueling increased malpractice insurance premiums. Critics also contend that injured parties may be left without adequate compensation because the attorney receives such a large percentage of the verdict. In addition, juries — realizing that attorneys' fees may absorb a significant portion of the award — tend to inflate their verdicts.

Proponents of the system maintain that it plays a vital role in the judicial process because the plaintiff often cannot afford the hourly rates of an attorney. Without the system, many plaintiffs with legitimate claims may be denied access to the courts merely because they are too poor to hire an attorney. Proponents also contend that the system does not encourage frivolous or multiple suits because plaintiffs' attorneys are not compensated if they lose a case taken on a contingency fee basis. Many plaintiffs' attorneys advance the expenses of litigation to their clients, and if the attorney loses the case, he or she may be unable to recoup the money advanced because of the plaintiff's inability to repay. Consequently, attorneys will not be inclined to accept cases on a contingency fee basis unless they are fairly confident of success.

Despite the arguments in support of the contingency fee system, it has been suggested that a sliding-scale fee schedule be devised to limit the percentage of the award given to an attorney in medical malpractice cases. With such a schedule, the contin-

gent fee percentage decreases as the amount of the award increases. Thus, more of the award is given to compensate the victim.

Such a system is not without controversy. Determination of an appropriate limitation on contingency fees is difficult. In order to induce attorneys to accept the risk of losing the money and time they expend in medical malpractice litigation, the potential fee must be sufficient to warrant the risk.

Several states, including Pennsylvania and New Jersey, have passed legislation to limit attorney's fees in medical malpractice cases. Under the Pennsylvania plan an attorney in a medical malpractice action may recover 30 per cent of the first \$100,000 awarded, and 25 per cent of any amount above \$200,000. In New Jersey, an attorney may recover 50 per cent of the first \$1,000, 40 per cent of the next \$2,000, 33 1/3 per cent of the next \$47,000, 20 per cent of the next \$50,000, and 10 per cent of any amount above \$100,000.

In Kansas, contingency fees are limited on the basis of their reasonableness. Generally, a contingency fee greater than 50 per cent is presumed to be unreasonable; however, it is the court's responsibility to determine if the actual fee in each case meets the reasonableness requirement.

Limitation on the Amount Recoverable

A few states responded to the crisis by enacting legislation that limits the amount recoverable in medical malpractice actions. The constitutionality of a cap or ceiling on damages has been continually challenged. While some courts have upheld this type of statute as constitutional, most have held these provisions unconstitutional.

The states that have held these provisions constitutional have found that limiting the amount of recovery in a medical malpractice case assists in assuring the availability of quality health care. For example, in 1980 in *Johnson v. St. Vincent Hospital, Inc.*, the Indiana Supreme Court held that a \$500,000 limit on recovery was constitutional. The court reasoned that if the cap had not been imposed, physicians would have refused to obtain malpractice insurance. Without malpractice insurance, injured patients would be unable to recover high damage awards which physicians could not afford to pay.

The jurisdictions holding ceilings on damages un-

constitutional have found that no nexus exists between a cap on damages and the availability of competent medical services. In *Carson v. Maurer*, decided in 1980, the Supreme Court of New Hampshire determined that the right to recover for personal injuries was an important substantive right. The court found that a cap limiting non-economic losses to \$250,000 was arbitrary and lacked a substantial relationship to the objective of the legislation. Consequently the statute violated the equal protection clause of the 14th amendment.

Similarly, in *Wright v. Central Du Page Hospital Association*, the Illinois Supreme Court struck down an Illinois statute that limited recovery in medical malpractice cases to \$500,000. The court determined that in order to constitutionally limit the amount recoverable in a medical malpractice action, the claimant would have to receive a *quid pro quo* in the form of lower medical costs. Since a ceiling on damages would not confer a *quid pro quo* to the claimant, the statute was arbitrary and violated the Illinois Constitution.

Conclusion

The medical malpractice crisis is a significant social issue in our society. Definite action must be taken to control the problem; otherwise, medical costs and insurance premiums for medical malpractice will continue to skyrocket. If this trend continues, health care providers may be forced to leave the field. This, in turn, could lead to less efficient medical services.

These concerns have led to legislative action in previous years, but more assertive legislative action may be necessary in the near future to control the medical malpractice dilemma. Physicians must do their part by providing quality medical care, utilizing effective peer review, and accepting personal responsibility for continuing medical education and appropriate consultation. Most importantly, the medical community must keep abreast of legislation that affects the medical profession and the stabilization fund. Each physician and health care provider should contact and encourage legislators to take forceful action to control the medical malpractice crisis.

Notes and references are available from Ms Wedel, 9800 Metcalf, Suite 510, Shawnee Mission KS 66212.



As ye sow—

We suspect that the volume of medical literature of the last thirty years relating to the legal, social, political, and economic aspects of medical practice exceeds that of all prior medical literature of all types. This issue of the *Journal* obviously adds to the phenomenon by presenting some current legal thoughts regarding the liability picture with particular orientation toward Kansas physicians.

Most such offerings enhance the physician's conviction that this picture is produced primarily by external pressures. We take a moment, therefore, to recall that some elements are well within the physician's personal province.

Communication: Tired as this is (both word and concept), it is still the best expression of the basic feature of the physician-patient relationship, and no one who claims the sanctity of that can ignore it. "Informed consent," although now represented in a legally approved document, should be nothing more than a formality if the informal personal involvement, so important to the patient, has been accomplished. Difficult as this may seem because of the complexity of modern medical service, the intrusions of other parties, the mobility of society, there is no adequate substitute.

Records: The importance of keeping accurate, complete, and current records regarding the patient's professional care is so trite as to excite annoyance that it should be brought up again. Nevertheless, the role of the record in the liability scene demands it. There is nothing that gladdens a plaintiff's attorney's heart more than finding an inadequate or incorrect record — unless it is one with obvious changes or corrections made after the fact. On the other hand, an honest, complete, straightforward record, even in the face of error, may well be the best protection available. But it is a professional necessity as well: by recording what is happening, what has happened and what should be done, it maintains the organization of the patient's care which can have only beneficial effects medically (and resolve potential problems with third parties).

Office: Apart from the potential risks of their services to patients, office personnel are a major factor in patient comfort. True, general attitudes may not lead to suits in themselves, but the atmosphere created by knowledgeable and understanding assistants can go far in encouraging the cooperation of patients and promoting the success of their care. The converse, of course, is true when patients feel unitized, homogenized, neglected, or just plain insignificant. A compassionate physician may survive inconsiderate assistants but compassionate personnel may well offset occasional lapses of physician behavior into an apparently unfeeling attitude. And not infrequently, patients will confide to receptive personnel things of potential significance they cannot bring themselves to tell the physician.

Professionalism: Obviously, appropriate professional preparation is essential for a healthy relationship — but the persistent recurrences of totally untrained persons posing as physicians demonstrates the frequent inability of the public (and sometimes physicians) to differentiate the real from the false. At the same time, it is testimony to the public's basic faith and dependence which individuals bestow on physicians. The academic qualifications, the years of preparation which physicians love to cite in their claims to distinction are the essential base but only the beginning. True professionalism goes beyond this to supplement and temper the fundamental principles to the patient — a tremendous obligation in its entirety. Although no physician can be all things to all patients, this effort approaches the *sine qua non* of a full and concerned medical professionalism.

Well, nothing new here. It has all been said before — many times. In putting it down on paper, though, we found that we had inadvertently come up with an acronym — CROP — unintended but not inappropriate since the points do reflect the medical sowing. But a gimmick was not our intent, even in this day when they are greatly celebrated. Certainly, no one should need it for its mnemonic value. Still, it might be a reminder from time to time. — D.E.G.



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Admitting Privileges: Who's to Judge?

A race is on. The win goes to the first party to tame industry's exploding health costs. The contestants are insurance companies, for-profit claims review companies, and physician peer review organizations. The prize is a major voice in determining the flow of treatment and money through the health care delivery system.

The same race is underway in the Medicare arena — with a sizable lead enjoyed, so far, by physician organizations. That lead stems purely from congressional mandate. Congress decided in 1982 that the public good is served by keeping medical decisions in the domain of local physicians.

Industry, although concerned with the public good, also owes allegiance to the bottom line. The contestant with the most efficient program, the most effective techniques, is the one that gets the job.

A technique that may separate the winner from the losers is called *pre-admission review*. It works like this. Before a physician admits a patient for elective hospitalization, his/her office telephones the review organization and gets certification of the need for hospital care. Reviewers use objective criteria for certification, the physician organizations distinguish themselves by the further use of professional medical judgement. Uncertified cases run a risk of not being reimbursed.

The good things about pre-admission review are that it is quickly implemented, reasonably inexpensive, and highly effective in improving the per-

centage of appropriate hospitalizations.

On the other hand, some physicians worry that pre-admission review might impede some emergency hospitalizations. Most physicians, however, realize that certification pertains only to elective admissions and, when conducted by a physician organization, carries a near vanishing risk to the patient. A far more frequent concern expressed by physicians is that the program is a bother and an infringement on the right to admit patients without question.

That concern is not shared by industry. Accustomed to audits, quality control inspections, and other accountability measures, industry now associates these practices with good business. And as health care costs exceed profits for many companies, hospital admissions are increasingly seen as candidates for scrutiny.

An unfortunate trait of pre-admission review is that almost anybody can get into the business. Whereas traditional review techniques required the cooperation of the local medical community, pre-admission review can be administered nationwide by anyone with a WATS line.

Control of hospital admissions is going up for sale. Let's hope Kansas physicians bid high — by supporting pre-admission review by their *own* peer review organization, the Kansas Foundation for Medical Care.

From The Kansas Foundation for Medical Care

Whoever Heard of "Kansas Medicine"?

Well, you have — now. And you'll be hearing of it — and from it — more and more in the future. After all, it shouldn't be totally unfamiliar since it represents an old friend, THE JOURNAL OF THE KANSAS MEDICAL SOCIETY, after a period of rehabilitation at a health farm.

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Forget the DRGs. This is in a class by itself. Watch for its release in January.

Law Students

(Continued from page 343)

the statutorily specified time, the order is considered final. If appealed, the court considers whether the law was applied correctly in the earlier hearing. The Kansas administrative process satisfies the due process requirement.

Workers compensation provides a viable solution to the liability vs. compensation-without-fault dilemma. The workers' compensation system can well serve as a model for the resolution of other similar societal issues. However, the facilitation of such innovative approaches would seem to require a familiarity with no fault compensation theories and terms. A recent Kansas approach to no fault is, of course, KSA 40-3109, which deals with personal injury protection (PIP) benefits in automobile insurance. Of significance to compensation without fault are the dram shop acts (such as Ill. Rev. Stat. 1953, Ch. 43, Sec. 1350), which place upon tavern operators an obligation to compensate the victims of drinkers. Is there a future in this concept for health care providers — a moving on of the law from tort fault liability to no fault compensation?

Acknowledgement

Dana M. Edwards, M.S.W., senior law student, assisted with the preparation of this article.

The Circle Closes

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Drug interactions: Aspirin, used concomitantly may decrease Motrin blood levels.

Coumarin: bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy or by nursing mothers.

Adverse Reactions: The most frequent type of adverse reaction occurring with Motrin is gastrointestinal of which one or more occurred in 4% to 16% of the patients.

Incidence Greater than 1% (but less than 3%)—Probable Causal Relationship

Gastrointestinal: Nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of GI tract (bloating and flatulence). **Central Nervous System:** Dizziness,* headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic/Endocrine:** Decreased appetite. **Cardiovascular:** Edema, fluid retention (generally responds promptly to drug discontinuation; see PRECAUTIONS).

Incidence less than 1%—Probable Causal Relationship**

Gastrointestinal: Gastric or duodenal ulcer with bleeding and/or perforation, gastrointestinal hemorrhage, melena, gastritis, hepatitis, jaundice, abnormal liver function tests. **Central Nervous System:** Depression, insomnia, confusion, emotional lability, somnolence, aseptic meningitis with fever and coma. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme, Stevens-Johnson syndrome, alopecia. **Special Senses:** Hearing loss, amblyopia (blurred and/or diminished vision, scotomata, and/or changes in color vision) (see PRECAUTIONS). **Hematologic:** Neutropenia, agranulocytosis, aplastic anemia, hemolytic anemia (sometimes Coombs positive), thrombocytopenia with or without purpura, eosinophilia, decreases in hemoglobin and hematocrit. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure, palpitations. **Allergic:** Syndrome of abdominal pain, fever, chills, nausea and vomiting, anaphylaxis, bronchospasm (see CONTRAINDICATIONS). **Renal:** Acute renal failure in patients with pre-existing significantly impaired renal function, decreased creatinine clearance, polyuria, azotemia, cystitis, hematuria. **Miscellaneous:** Dry eyes and mouth, gingival ulcer, rhinitis.

Incidence less than 1%—Causal Relationship Unknown**

Gastrointestinal: Pancreatitis. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities, pseudotumor cerebri. **Dermatologic:** Toxic epidermal necrolysis, photoallergic skin reactions. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Bleeding episodes (e.g. epistaxis, menorrhagia); **Metabolic/Endocrine:** Gynecomastia, hypoglycemic reaction. **Cardiovascular:** Arrhythmias (sinus tachycardia, sinus bradycardia). **Allergic:** Serum sickness, lupus erythematosus syndrome, Henoch-Schönlein vasculitis. **Renal:** Renal papillary necrosis.

*Reactions occurring in 3% to 9% of patients treated with Motrin. (Those reactions occurring in less than 3% of the patients are unmarked.)

**Reactions are classified under "Probable Causal Relationship (PCR)" if there has been one positive rechallenge or if three or more cases occur which might be causally related. Reactions are classified under "Causal Relationship Unknown" if seven or more events have been reported but the criteria for PCR have not been met.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine so alkaline diuresis may be beneficial.

Dosage and Administration: Rheumatoid arthritis and osteoarthritis. Suggested dosage is 300, 400, or 600 mg t.i.d. or q.i.d. Do not exceed 2400 mg per day. Mild to moderate pain: 400 mg every 4 to 6 hours as necessary.

Caution: Federal law prohibits dispensing without prescription.

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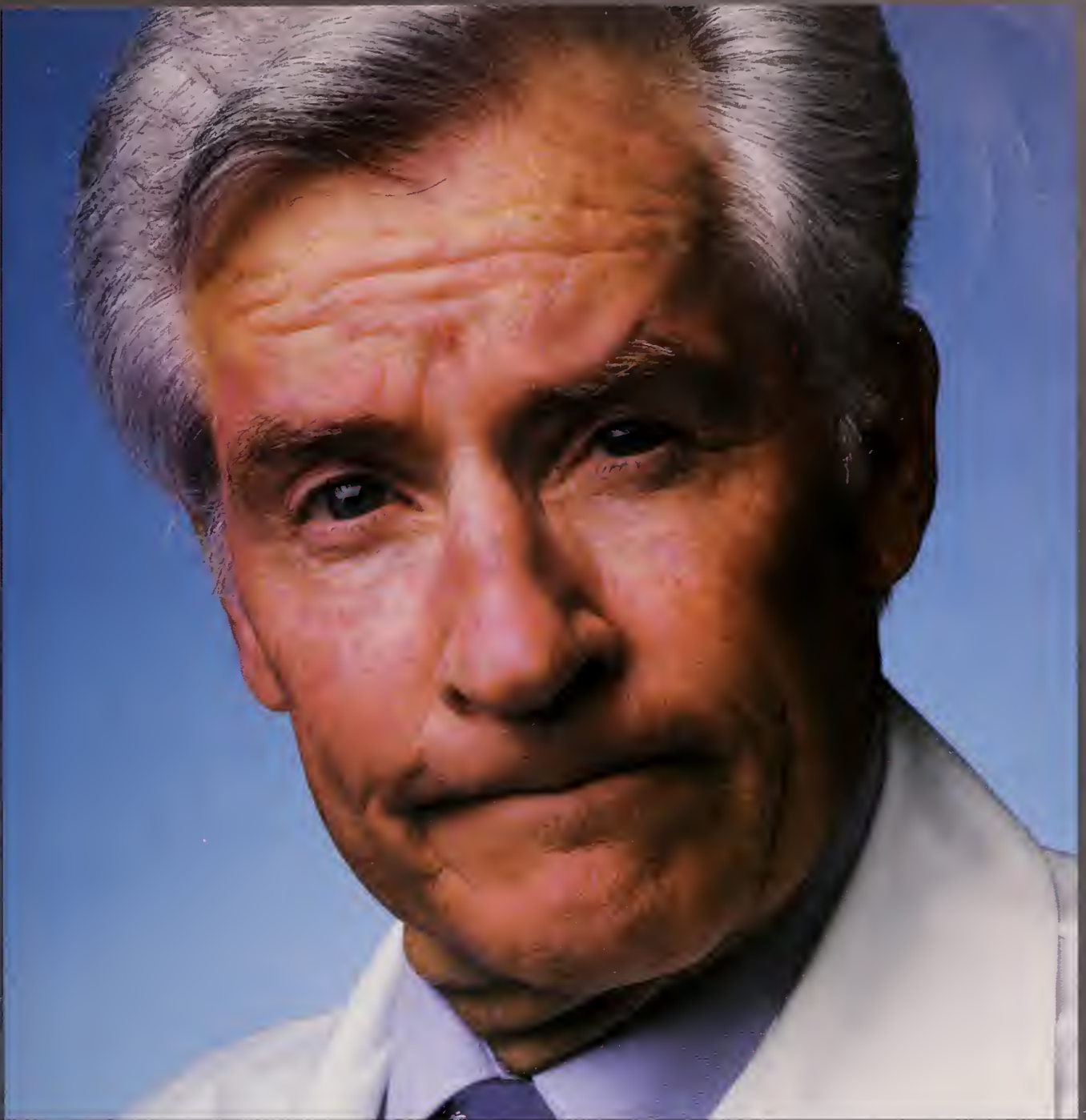
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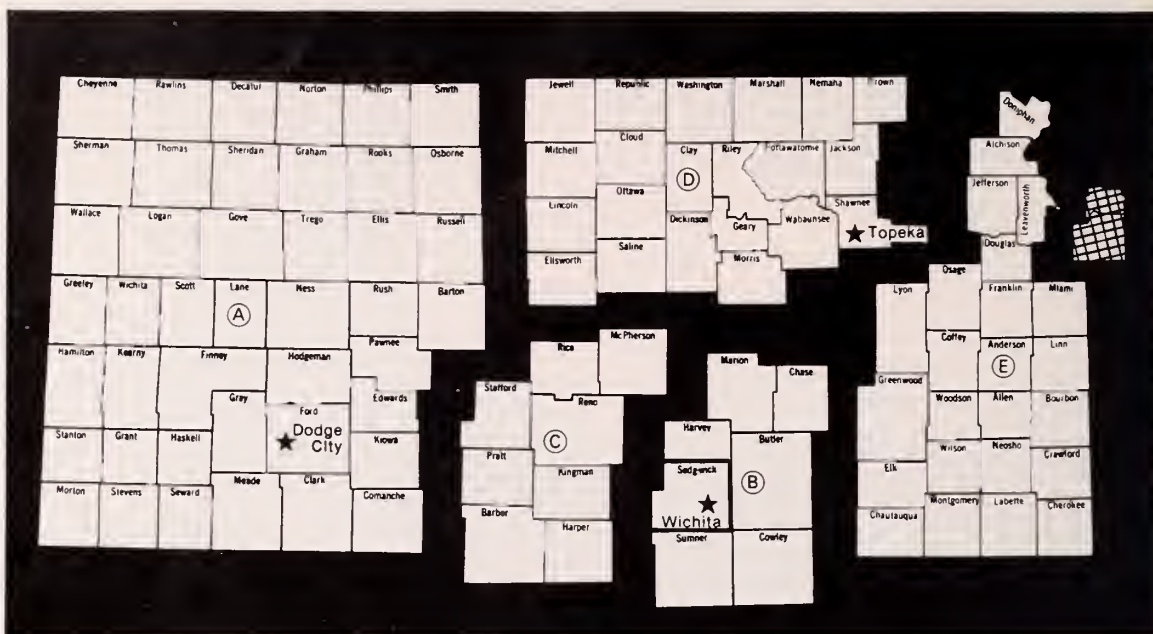
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Permanent Resolution

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other tort reforms. The program that has been outlined by the Medical Society for the upcoming legislative year should be supported by all physicians, not only with money but also with time and effort. Nevertheless, the medical profession must look beyond 1985 for a more permanent and lasting solution to this continuing problem that has plagued physicians for the past 20 years.

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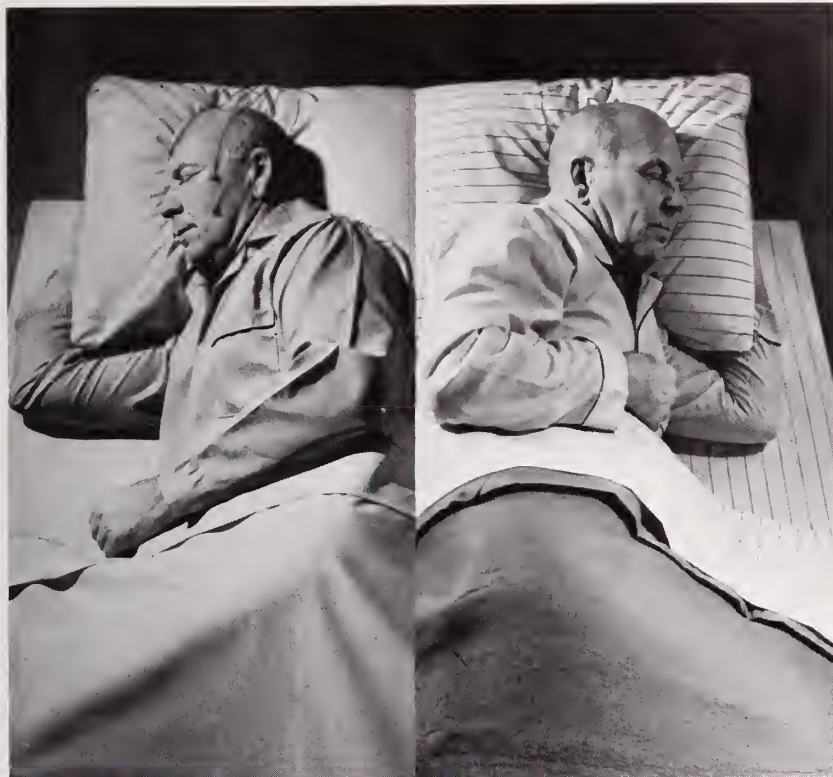


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